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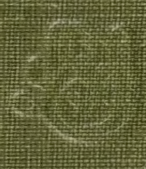

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POLIOMYELITIS

WITH ESPECIAL REFERENCE TO THE TREATMENT

BY

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PREFACE.

In spite of the fact that poliomyelitis has not appeared in epidemic form during the last ten years, the interest of the medical profession in this disease has by no means diminished. In truth, there have been sufficient sporadic cases to indicate that another outbreak of the disease in extensive form is not unlikely. In view of this possibility, it seems well to have at hand a modern, comprehensive account of the disease—one that presents all its phases, but in particular gives a clear understanding of the principles of treatment. Such a work answers the need of the general practitioner, who controls many factors that influence the ultimate recovery of function, and, with its complete delineation of medical and surgical procedures, serves also as a means of reference for the orthopedic surgeon.

Since the last extensive epidemic, in 1916, the study of poliomyelitis has been diligently pursued. Some of the work has been of a negative character, some of it will require verification, and some of it has been based upon faulty methods. A crystallization of the most valuable observations shows that an advance has been made in our study of the disease. We have a wider knowledge of the clinical manifestations; we have a clearer understanding of the pathological process; and we have more accurate information regarding the specific nature of the disease. The greatest progress has been made in the methods of treatment, and particularly in the orthopedic field. Deformity can now be prevented in nearly every instance; the return of function can be hastened by proper treatment of the involved area; and, finally, by the use of surgical procedures, the wearing of cumbersome apparatus may be obviated in the majority of cases.

From the voluminous material that has been published on poliomyelitis, the writer has tried to select the factors that are recognized as having definite value. In the epidemic and experimental field, in which much remains to be determined, the most recent investigations have been reported. From the large number of operative procedures that have been devised for paralytic conditions, the writer has selected those measures that have stood the test of time and can be accepted as standard.

In the preparation of this work, a tremendous number of publications extending over a twenty-year period have been read, and their value carefully weighed. A painstaking effort has been made to compile a bibliography that is both comprehensive and usable.

The writer acknowledges with gratitude his indebtedness to Dr. Harry C. Solomon, Dr. P. Somers Smyth, Dr. Charles R. C. Borden, and Dr. William L. Aycock, who have read and helpfully criticized certain chapters of the manuscript. Particular thanks is due to colleagues who have permitted reproduction of their operative measures, and who have supplied cases for illustrative purposes. The writer is especially grateful to Miss Helen Bixby, who has diligently assisted in the preparation of this work.

W. R. M.

Boston, 1927.

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POLIOMYELITIS.

PART I.

ETIOLOGY, SYMPTOMS, DIAGNOSIS AND PROGNOSIS.

CHAPTER I.

HISTORY.

Unlike most diseases of epidemic character, which may be traced historically in literature to very early times, poliomyelitis is practically unmentioned up to the year 1840. If the disease was prevalent, the outbreaks were not sufficiently extensive or localized to attract attention.

From time to time, writers have cited ancient cases, but as they were based on very slight evidence, it is possible that some of them were due to lesions other than poliomyelitis. Mitchell[1] reported finding a shortening of the femur in the skeleton of an Egyptian mummy, which he attributed to infantile paralysis. As no other evidence of this disease was present, the condition might equally well have been of congenital origin. In II Samuel, 4:4, there is mentioned the case of Mephibosheth. "And Jonathan, Saul's son, had a son that was lame of his feet. He was five years old when the tidings came of Saul and Jonathan out of Jezreel, and his nurse took him up, and fled: and it came to pass, as she made haste to flee, that he fell, and became lame."

In the Letters of Madame de Maintenon,[2] we find mentioned that one of the sons of Louis XIV suffered from a sudden illness, followed by paralysis. Sir Walter Scott was disabled in childhood after a moderately severe attack of paralysis of one leg. Deformities such as result from infantile paralysis have also been depicted in paintings and sculptures.

The first description of poliomyelitis recorded in medical annals is found in Underwood's[3] *Treatise on the Diseases of Children*, published in 1793, in which he says, "The disorder intended here is not noticed by any medical writer within the compass of my reading. or is not so described as to ascertain the disease." Underwood did not appreciate the true cause of the illness, but he believed it arose from debility. He noted that it occurred usually in the lower extremities. If only one limb was affected, Underwood recommended blisters or caustics on the os sacrum and greater trochanter, and volatile and stimulating applications to legs and thighs. When both legs were paralyzed, he advised irons to support the limbs.

Shaw,[4] in his publication *Nature and Treatment of Distortions*, connected the disease with weaning. Jörg,[5] Bartsch,[6] Brück,[7] and others also described the disease, but their reports are of little value.

The next important record was made by Badham,[8] of Workrop, England. His clinical picture was more complete than Underwood's in that it included the cerebral symptoms, and called attention to the occurrence of the disease in childhood.

The first systematic study of the disease was made by Jacob von Heine[9] of Cannstadt, Germany, a report of which was published in 1840 in his famous monograph *Observations on Paralytic Conditions of the Lower Extremities and Their Handling*. His account, based on a personal observation of 21 cases, outlined clearly the important features of the disease, including etiology, pathology, diagnosis, prognosis, and treatment. Jacob von Heine was the only one of the early observers to recognize that the pathological process attacked the spinal cord, but he had no microscopic proof to substantiate his hypothesis. Somewhat later, his theory was proved to be correct, and in the second edition of his monograph, published in 1860, he termed the disease "Infantile Spinal Paralysis." Von Heine also noted the acute febrile period preceding the paralysis, and suspected the contagiousness of the disease. For treatment, he recommended baths and exercises, for which he devised a simple apparatus. He also described various surgical principles.

From 1840 to 1860, little knowledge was contributed on any phase of the disease. Kennedy,[10] West,[11] Chassaignac,[12] and McCormac[13] mentioned the malady in their writings, but their descriptions were vague.

About 1860, more attention was given to the study of the pathology of poliomyelitis. Before 1863, only 5 autopsies had been performed, due undoubtedly to the fact that the disease was seldom fatal, and that there was no opportunity to make pathological investigations while paralysis predominated. In these early autopsies, no spinal lesions were recognized, with the result that an erroneous opinion developed as to the pathology (Jacobi[14]).

Cornil[15] was the first to describe pathological changes in the spinal cord, but while he noted the atrophied condition, he failed to recognize the important feature, the destruction of the anterior horns. A few years later, Prévost and Vulpian[16] called attention to the affection of the anterior cornua. Several theories were advanced as to the origin of these lesions. Some investigators believed that they were due to congestion, and others that they were caused by primary sclerosis. The true significance of these changes of the spinal cord was first appreciated by Charcot,[17] who associated the atrophy of the ganglion-cells in the anterior horns with the muscle atrophy. Further study of the pathological anatomy was made by Lancereaux and Pierret,[18] Leyden,[19] Schultze,[20] Goldscheider,[21] and others.

In the more recent studies it has been shown that not only all parts of the central nervous system may be involved, but that the lesions may be scattered throughout other structures, particularly in the lymphatic system and the parenchymatous organs. The changes in these structures will be discussed in detail in the Chapter on Pathology.

Meantime, efforts were being made to establish the etiology of the disease. Its infectious nature was first shown clinically, in 1884, by Strümpell.[22] An understanding of the true character of the disease, as it is known today, began with Medin's[23] study, made during the first epidemic of any size, which occurred in 1887 at Stockholm. Wickman's[24] contributions on this phase of the disease also are important. In his study of the Swedish epidemic of 1905, he discovered that many cases are not associated with paralysis. The clinical evidence evincing the infectious nature of the disease was corroborated by many experimental findings, which will be referred to in a later chapter.

About 1893 began the period of experimental research in the endeavor to discover the infectious organism. Dauber,[25] Goldscheider,[21] and Siemerling[26] examined the cords of patients who had succumbed to the disease, but failed to find pathogenic bacteria. In 1898, Bülow-Hansen and Harbitz[27] found a number of minute corpuscles stained deep red by Loeffler's method, after incubation of an active Berkefeld filtrate bouillon. Monkeys inoculated with this filtrate became paralyzed. In 1909, Leiner and von Wiesner[28] in Vienna, Flexner and Lewis[29] in New York, Landsteiner and Levaditi[30] in Paris, and Römer,[31] Kraus,[32] and Meinicke[33] in Germany, working independently, announced that the agent belonged to the group of filterable viri. In 1913, Flexner and Noguchi[34] reported that they had isolated an organism from the spinal cord, which fulfilled Koch's requirements. Further experimental work is needed to confirm their discovery, before this microörganism may be considered as the established cause.

Attempts were then made to establish artificial immunity. Flexner and Lewis[29] found that monkeys inoculated with serum derived from a recovered case would not become paralyzed. Netter, Gendron, and Touraine,[35] and Netter and Salanier[36] soon tried this method of immunity in humans. Such experiments are being carried on today. This subject will be treated more fully under the section on serum treatment.

Much progress has been made in the treatment of poliomyelitis since the time of Heine, when caustics and crude apparatus were used. Massage, muscle training, modern apparatus, and especially operative procedures are essential measures in the treatment at the present time.

The importance of the prevention of the development of deformities, and the necessity of their early correction, when they occur, are gradually becoming more fully appreciated. Once the value of these principles is impressed on the minds of all surgeons, the number of deformities in infantile paralysis will be almost negligible. The possibility of the restoration of function to paralyzed limbs is greater today than ever before, due to the therapeutic and operative measures at our disposal.

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CHAPTER II.

EPIDEMIOLOGY.

THE occurrence of poliomyelitis in epidemic form has been noted only within recent years. It is probable that outbreaks did occur in earlier times, but undoubtedly they were overlooked, or were not reported, or were confused with epidemics of other diseases that have features in common with poliomyelitis.

The first appearance of the disease in epidemic form seems to have been in 1858 in the Scandinavian Peninsula. Gradually the outbreaks assumed significant proportions, until in 1905, the disease occurred in pandemic form in Europe. The gradual development of poliomyelitis from sporadic to pandemic form is similar throughout all countries, as may be noted in the historical résumé which follows.

We can only theorize as to the factors that account for the tendency of the disease to spread and to increase in severity. The fluctuation in the virulence of the microörganism may be responsible. Flexner[1] has demonstrated experimentally that the passage of the infectious organism from monkey to monkey leads to an increase in virulence. Then again, outbreaks may occur only when there is a sufficiently fertile field, as in the case of a community that has not become relatively immune by exposure to the disease, or where there are many young children.

The increasing frequency of poliomyelitis in epidemic form has aroused widespread interest. Statistics have been compiled of the outbreaks that have occurred throughout the world. Extensive study has been made, and still is being carried on, to discover the factors common to all epidemics, and to account for the transmission of the disease. In the following pages, the writer will sum up the general conclusions reached by the investigators of the different epidemics.

THE PREVALENCE OF EPIDEMICS THROUGHOUT THE WORLD.

No country seems to have escaped the ravages of this disease. It has occurred under extremes of latitude, and under varying climatic conditions. The most extensive outbreaks have taken place in the northern parts of Europe and America. The development of the power of epidemicity in this disease was first noted in Europe, and the western hemisphere was not affected until several years later.

The number of cases in each epidemic can be estimated only approximately. In many countries, the morbidity has been based upon the mortality-rates, which often include the report of deaths caused by diseases other than poliomyelitis. For instance, in the United States, prior to 1910, deaths from infantile paralysis were reported among fatalities from other diseases of the nervous system.

Infantile paralysis was not made reportable in any country until recent years. Norway listed poliomyelitis among notifiable diseases in 1904, Sweden in 1905, and England in 1911. Even since the time when the report of cases was made obligatory, the morbidity rates have not been accurate. In many countries, notification is not uniformly compulsory. Registration is often faulty; errors occur in diagnosis; many slight attacks are unobserved even in times of epidemics; and physicians fail to report cases. These factors must be kept constantly in mind in considering the number of cases in the epidemics recorded below.

During and since the World War, we have been unable to procure reliable foreign statistics from many countries. When possible, the writer has secured personal information in regard to the general trend of the disease since 1914. It will be noted that the statistical data in the following countries is not complete: Norway and Sweden, Denmark, Austria, Holland, and Russia.

In the preparation of the list of epidemics, the writer has consulted the compilations of Ruhräh and Mayer.[2] Lavinder, Freeman, and Frost,[3] Wickman,[4] and Low;[5] the Monograph issued by the New York Health Department of 1917;[6] and the Public Health Reports issued by the United States Public Health Department. References quoted in full in these compilations are not repeated in the bibliography at the end of this chapter; simply the authority is mentioned. The sources of new material are given in detail.

The authenticity of the earliest account of poliomyelitis, which was reported by Colmer, in 1843 is often questioned, as it is based on hearsay evidence. Colmer wrote, "Whilst on a visit to the parish of West Feliciana, Louisiana, in the fall of 1841, my attention was called to a child, about a year old, then slowly recovering from an attack of hemiplegia. The parents (who were people of intelligence and unquestionable veracity) told me that 8 or 10 other cases of either hemiplegia or paraplegia had occurred during the preceding three or four months within a few miles of their residence, all of which had either completely recovered, or were decidedly improving.

The little sufferers were invariably under two years of age, and the cause seemed to be the same in all—namely, teething."

There is little doubt, however, that the next record, covering an epidemic which occurred fifteen years later in Norway, dealt with a true outbreak of poliomyelitis. A. C. Bull reported the details of this epidemic to the State Medical Board. It comprised 14 cases, among which were 2 of the abortive type, which had a short fever, but only indistinct pareses. Bull also questioned the contagiousness of the disease (Leegaard).

No other record of an epidemic is found in literature until 1881. Beginning with that year, the outbreaks became more frequent, and occurred throughout the entire world. The epidemicity of the disease in the different countries may be outlined as follows:

Norway and Sweden.—Poliomyelitis first appeared in epidemic form in Scandinavia. Before the year 1905, the data of the prevalence of the disease is inaccurate, for in some years the deaths were reported among those due to cerebrospinal meningitis. In 1881, Bergenholtz reported that he had observed a small epidemic of 13 cases in Umea, a town of northern Sweden. In 1886, Oxholm described a group of 9 cases, which occurred at Mandel.

From that year up to 1905, several small epidemics were reported. Most important among them were two epidemics in Sweden, both at Stockholm, one in 1887, and the other in 1899, and two epidemics in Norway, one of 64 cases at Hvaler (Nannestadt), and the other at Bratsburg (Leegaard). Medin made an extensive study of the first epidemic. Leegaard studied carefully the epidemic at Bratsburg, and pointed out the spread of the disease along the line of travel. He also called attention to the short duration of the incubation period.

Smaller outbreaks were recorded as follows:

1886— 9 cases at Mandel, Norway, reported by Oxholm.
1888—14 cases at Ammeberg, Sweden, reported by Larsson.
1895—21 cases at Stockholm, Sweden, reported by Medin.
1903—20 cases at Göteberg, Sweden, reported by Wickman.
1903—19 cases at Hadeland and Land, Norway, reported by Gierswold.
1904—20 cases at Aafjorden, Norway, reported by Platou.
1904—41 cases at Fredrichstad, Norway, reported by Low.

Between the years 1904 and 1908, poliomyelitis was widely diffused throughout the Scandinavian Peninsula. In 1905, the disease had spread over many parts of Sweden, and for the first time was made notifiable. Fairly complete records have been compiled since that year. The most extensive epidemic yet recorded occurred in southern Sweden in 1905; it comprised 1199 cases, with 145 deaths. Its distribution was irregular, the rural districts being ravaged, and the cities spared. Wickman investigated thoroughly 1025 cases in this epidemic, giving special attention to the possibility of contagion, the frequency of abortive cases, and the meningeal symptoms. He was the first to recognize that the disease is conveyed from person to person, and that the abortive as well as the paralyzed case may spread the affection.

During the same year, the disease was prevalent throughout Norway. Harbitz and Scheel reported an outbreak of 719 cases.

In 1906, the disease was still present throughout the Peninsula, as shown by a total of 407 cases reported in Norway (Leegaard), and 429 cases in Sweden (Kling, *et al.*). Wickman reported an epidemic of 50 cases at Smedjebacken, Sweden. The northern part of Norway suffered more than the southern; 347 cases were reported at North Trondhjem, 168 at Nordlands, 199 at Tromsö, and 60 at Finmarken. The rural districts suffered more than the towns.

The disease continued to occur sporadically during the next four years, a total of 671 cases, 376 cases, 229 cases, and 237 cases being reported for the years 1907, 1908, 1909, and 1910 respectively (Leegaard; Kling, *et al.*).

In 1911, a most extensive epidemic occurred. Leegaard reported 1158 cases in Norway, and Kling reported 3840 cases in Sweden. An interesting relationship was noted in Sweden between the incidence of the disease and the density of the population; 3 per cent of the rural population was attacked, whereas only 1 per cent of the whole population developed the disease. The large cities, however, showed a greater incidence than in the epidemic of 1905. For instance, in Jönköpings, of a population of 216,000, only 55 cases were recorded in 1905, whereas 1065 were reported in 1911. Multiple attacks in families were noted. Abortive cases comprised 33 to 50 per cent of the entire number. The mortality varied from 10 to 26 per cent.

The high prevalence continued throughout Sweden in 1912; Kling, Pettersson, and Wernstedt recorded 1458 cases between January 1st and August 15th, and at least 3344 cases were reported in the second half of the year (Low). In Norway, the disease continued to be prevalent, 425 cases with 74 deaths being recorded.

In 1913, the number of cases diminished, 1125 occurring throughout the entire year in Sweden, and 543 in Norway.

Denmark. The first record of the disease in this country included isolated cases, which occurred in 1903 and 1904. Coincident with the epidemics in 1905 in Norway and Sweden, the number of cases in Denmark increased, and 100 were recorded. But few cases were reported from 1905 to 1909. In 1910, there were three outbreaks, localized at Helsinger, Frederikssund and Vestervig. A total of 190 cases were recognized, among which were 7 deaths.

During the next two years, 1911 and 1912, the disease was more prevalent. In 1911, 301 cases occurred, and the mortality-rate was 10.3 per cent; in 1912, 371 cases with a mortality-rate of 13.5 per cent were reported.

In 1913, there was a marked reduction in the number of cases, 183 being reported. In 1914, 140 cases occurred (Low).

Germany.—In the eighties and the nineties, Germany suffered several minor outbreaks, including one of 5 cases in 1889 at Thuringia (Briegleb), one of 4 cases in 1898 at Kiel (Von Pleuss), one of 15 cases in the same year at Frankfort-on-the-Main (Auerbach), and another outbreak of 5 cases in 1899 at Thuringia (Briegleb).

It was not until 1908 that the disease began to attract special attention, when one outbreak of 50 cases was noted at Marburg (Römer and Müller), and another of 36 cases at Heidelberg (Hoffman).

In 1909, serious epidemics visited Germany, involving some of the most densely populated industrial centers, especially Westphalia, Hesse-Nassau, Pomerania, Hanover, Breslau, and Schleswig-Holstein (Reckzeh, Müller, Peiper, Eichelberg, Forster, and Myer). It was estimated that nearly 2000 cases had occurred up to the end of September, 1909 (Netter). Müller, who observed a large number of cases, stated that 75 per cent were under four years of age. The largest focus was in Westphalia where about 500 cases were reported. Krause, Reckzeh, and others studied this outbreak. It appears to have been of unusual virulence, as the mortality-rate was about 18 per cent. Faucial inflammation was an initial symptom in many cases. According to Krause, there were intestinal symptoms in 90 per cent of the cases, an observation which led him to believe that the virus entered the body by the intestinal tract. A coincidental paralytic disease was noted among fowl.

The disease continued prevalent throughout Germany during the next four years. In 1910, an epidemic ravaged Schleswig-Holstein. In 1912, Uffenheimer reported the occurrence of 197 cases in Bavaria. In 1913, two outbreaks were recorded, one of 71 cases at Heidelberg (Lust and Rosenberg), and the other of 168 cases at Hesse (Langermann).

In 1923, there were 550 cases. In 1924, 507 cases were reported, the incidence being highest in Mecklenburg-Schwerin. Three hundred and eighteen cases occurred in Prussia, Magdeburg having the greatest number.[7]

Austria.—The record of epidemics in Austria begins with an outbreak of 42 cases in 1898 at Vienna (Zappert). The annual average in the preceding ten years had been 12 cases. The disease continued to prevail in sporadic form from 1898 to 1908. Neurath recorded 96 cases for the years 1902 to 1909.

In 1908 and 1909, the small outbreaks culminated in a serious epidemic totaling over 1000 cases. The largest outbreak, of 555 cases, occurred in Vienna and lower Austria, and was carefully investigated by Zappert and a collective investigating committee. Although the cases showed a tendency to occur in groups, Zappert was unable to prove the contagiousness of the disease. The distribution of the cases was very irregular, and the case incidence showed no variance according to the density of the population. Four hundred and thirty-four cases occurred at Steiermark, with a mortality of about 13 per cent (Potpeschnigg). Smaller foci appeared at Steyr (Lindner and Mally), Löcker, Linz, and Urfahr (Stiefler), and at Kärnten (Puschnig).

The disease continued prevalent throughout 1910, a total of 158 cases being reported. In 1911 the number decreased considerably. The incidence again increased in 1912 and 1913, 117 cases and 127 cases being reported for the respective years. In the first half of the year 1914, 56 cases occurred.

France.—It is difficult to estimate even approximately the number of epidemics in France. There has been no compilation of statistics. The disease was not made notifiable until 1911, and figures from even that year are by no means accurate, since the Government has not made notification obligatory.

France seems to have suffered no serious epidemic, although small outbreaks have been reported since the year 1885. Cordier made a careful study of a small epidemic which occurred in 1886 at Saint Fay-l'Argentière. Thirteen cases broke out among the 1400 or 1500 inhabitants, and 4 of those affected died. Invariably there was a record of direct or indirect contact with an acute case. Many of the children were breast-fed. It is worthy of note that Cordier suggested that the disease might be of infectious origin.

Other small outbreaks recorded were:

1893— 9 cases at St. Girons (André).

1906–10—69 cases at Toulouse (Bezy and Sorrel).

1913—18 cases at Leblanc Azay (Veluet).

1914—13 cases at Flers de l'Orne (Guinon and Pouzin).

The largest outbreaks occurred in 1909 at Paris and in 1911 at Pologne. Netter, who has given much time to the investigation of poliomyelitis, recorded the incidence of the Paris epidemic at 100 cases. Mme. Biehler recorded 166 cases in the epidemic at Pologne.

The writer has not been able to obtain any report of the prevalence of the disease during the period of the World War. Inquiries sent recently to several pediatrists in France, who specialize in nervous diseases, have disclosed that at the present time there are several endemic foci in France. No part of the country appears to be immune (Lamy[8]).

Belgium.—Low[5] reports that Belgium has not escaped the disease. In the *Revue Médicale de Louvain,* Ide stated that it was his opinion that Belgium suffered as much in proportion to her population as Germany and France. There are no details as to the number of cases or their distribution.

Holland.—From 1906 to 1913, sporadic cases of poliomyelitis were reported to have occurred throughout Holland (Starke, Schippers, and DeLange). There were records of only two definite outbreaks, one of 24 cases in 1909 at Leyden (Krause), and another of 14 cases the same year at Warmsweld and Zutphen (Krause). In 1912, Schippers and DeLange recorded a number of cases which they had treated in Amsterdam. In the following year, cases were reported in five localities, comprising Rotterdam, Klasswaal, Mynsheerenland, Wehl, and Empel, but the exact number of cases was not ascertained. In 1914 there was a small epidemic at Rotterdam.[9]

Switzerland.—Switzerland presents a record similar to that of Holland. Isolated cases were observed at the hospital at Basle between the years 1885 and 1909 (Hagenbach). Eichhorst[10] reported a group of cases at Zurich between September and November, 1909. In 1910, an outbreak of 37 cases occurred in East Switzerland (Brandenberg). Dubois[11] estimated the number of cases from 1913 to 1922 at 552. In the first part of the year 1923, 123 cases occurred.

Italy.—The disease has prevailed in Italy since 1883. Although the infection has shown a tendency to increase, there is no record of a large epidemic. During the year 1909, one outbreak of 62 cases was reported in Adriatic Italy (Gioseppsi), and another of 60 cases in Florence and vicinity (Mayanz). Up to that time reports had been made of the following epidemics:

1883— 5 cases at Arenzano (Buccelli).
1895— 7 cases at Montespertoli (Pieraccini).

1895—17 cases at Revecca (Buccelli).
1897—22 cases at Conegliano (Fabis).
1899— 5 cases at Valley of Arno (Simonini).
1903—26 cases at Parma (Lorenzelli).

In the official report for 1913 on the "Statistica delle Cause di Morte," published at Rome in 1915, deaths from poliomyelitis were as follows:

1904, 228; 1905, 293; 1906, 322; 1907, 330; 1908, 341; 1909, 333; 1910, 364; 1911, 344; 1912, 339; 1913, 379.

During the period from 1910 to 1920, the disease continued to occur mostly in sporadic form, or in very small epidemic foci. Simonini[12] collected statistics of 3411 cases which occurred in this decade throughout Italy.

British Isles.—The disease is recorded as having been endemic throughout England since 1896, when Pasteur reported 7 cases at Much Hadham. In 1898, Buzzard lectured on the infectious origin of infantile paralysis and described a number of cases which he had observed in London and elsewhere. London suffered outbreaks in 1897, 1904, and 1909. From 1910 to 1914 poliomyelitis was more prevalent. The largest outbreaks were:

1909—37 cases at Bristol (Parker).
1910—33 cases at Carlisle (Beard).
1910—37 cases at Barrow-in-Furness (Garrow).
1910—62 cases about Edinburgh (Low).
1913–14—37 cases at Tyrone, Coagh Co. (Burgess).
1913—24 cases at Belfast (Low).
1913—56 cases at Barrow-in-Furness and Westmorland (Low).

Platt,[13] in an article published in 1924, writes, "In the British Isles, the number of cases occurring in sporadic form, and in small groups, continues to rise."

Australia. Altman reported an outbreak of 14 cases of poliomyelitis in 1895 at Port Lincoln. This was the first account of the disease in epidemic form in Australia, although sporadic cases had occurred before this time.

In 1903 and 1904, during the summer months, a somewhat extensive epidemic occurred in Australia, with four main foci: The first appeared at Queensbergen, New South Wales (Blackhall); the second at Sydney (Litchfield); the third, of 34 cases, at Stanmore (Wade); and the fourth and largest, of 108 cases, at Brisbane (Ham). Wade found a number of instances of more than one case in a family, some of whom suffered from the so-called abortive form. In 1908, Victoria was the center of an epidemic of 137 cases, with a fatality rate of 4.5 per cent (Stevens). Small outbreaks were recorded during the years 1909, 1910, and 1911.

In 1912, the disease was made notifiable in New South Wales. Twenty-eight cases were reported in Sydney and its suburbs. In 1913, about 43 cases occurred in New South Wales, of which 22 were in the Metropolitan District of Brisbane.

According to the annual report for 1914 of the Director-General of New South Wales, there were 78 cases throughout the State, 63 of which occurred in the Metropolitan District of Sydney. Statistics showed that the number of cases of poliomyelitis was increasing in this section of Australia. In the latter half of 1914, an epidemic comprising 207 cases with 29 deaths occurred in the Brisbane Metropolitan District. Up to June 30, 1915, 112 cases had been reported throughout the other sections of Queensland.

Epidemics also occurred in 1915 and 1916, reaching their height in the second half of the summer and in the autumn. There was then an interval until 1921, during which only isolated cases were reported. In 1921, 1923, and 1924 there were mild epidemics, 236, 136, and 229 cases being reported for the respective years. No outbreaks were reported in 1925 (Dick[14]).

New Zealand.—The disease did not begin to appear in fatality statistics until 1908, when 3 deaths were reported. In 1914, an epidemic of 268 cases occurred. Only 10 cases were reported during 1915 (Low).

In 1925, a serious epidemic broke out in New Zealand. Available data up to May of that year disclosed that 970 cases had occurred (Dick[14]).

Islands in the Western Pacific.—*Nauru.*—In 1910, an epidemic attacked the 700 inhabitants of this island. The disease occurred in mild form, as only 38 deaths were recorded, and only 50 individuals remained paralyzed after three months. The origin of the epidemic was never discovered (Müller).

Hawaii.—In 1910, the disease was prevalent in epidemic form in this island, and it has since occurred in sporadic form in 1913 and 1914 (Low).

Russia.—Three epidemics have been reported in this country:

1905—15 cases at St. Petersburg (Jogichess).

1908—49 cases at Russia—a village (Schwarz).

1909–10—29 cases at St. Petersburg (Jogichess).

Canada.—The disease has been endemic in Canada for a number of years, but no large epidemic has ever occurred (Russel and

Hodgetts). It is difficult to obtain statistics of the number of cases, as previous to the year 1920 no organized effort was made to collect vital statistics in Canada. In the provinces in which statistics were compiled, poliomyelitis was listed among seventy-three other diseases of the spinal cord. In 1924, the first attempt was made to collect records of the number of cases of poliomyelitis; 217 cases were reported (E. S. Macphail[15]).

Cuba.—The epidemic which occurred in 1909 in Cuba is of interest, as it was the first to be reported from the tropics. The disease had probably existed previously in Cuba, but only in sporadic form, with low case-incidence. In 1907 and 1908, the cases became more numerous in the neighborhood of Havana, and in 1909, an epidemic of about 140 cases raged in the province of Santa Clara. There was not a single instance of multiple cases in one family. Lebredo and Recio, who described the epidemic, questioned if the virus may not have been imported from the United States into Havana, and thence to the interior of the island. The fatality was only 7.89 per cent.

Jamaica.—Only scanty information has been collected on the prevalence of the disease in this country. In 1913 and 1914, it was present in sporadic form. In 1915, there were 6 cases (Low).

South American Countries.—No serious epidemics have been reported from South America. An outbreak occurred in 1909 at Sao Paulo, Brazil; in 1911, 39 cases were reported in Buenos Aires, Argentina. The Annual Report of the Surgeon-General of the United States Public Health Service for the fiscal year 1912 and 1913, states that the number of poliomyelitic cases appears to be increasing in Ecuador.

Alaska.—This country suffered an epidemic of 30 cases in 1913. It was noted especially that the cases were more numerous in localities where dogs and flies were abundant (Pierson).

United States.—The United States presents a history similar to that of European countries, in that small outbreaks occurred before severe epidemics.

Macphail and Caverly recorded the first epidemic in the United States, which occurred in 1894 at Rutland, Vermont. They were unable to ascertain the exact number of cases, but they estimated at least 130. Most of the cases were children, but a few adults were affected. A paralytic disease was noted among animals, especially among horses. Although postmortem examinations of these animals showed cord lesions, there was no evidence that the disease was poliomyelitis.

Other small outbreaks which occurred in the nineties in the United States are as follows:

1894—10 cases at North Adams, Massachusetts (Brackett).
1896—15 cases at Greene County, Alabama (Bondurant).
1896— 9 cases at Cherryfield, Maine (Taylor).
1896— 8 cases at San Francisco and Napa, California (Sherman).
1897—12 cases at New York City (Taylor).
1899—30 cases at Dutchess County and Poughkeepsie, New York (Mackenzie).
1899— 4 cases at San Joaquin Valley, California (Newmark).

During the next decade, 1900–1910, there was a great increase in the size and severity of the epidemics. At the beginning of the period, two outbreaks were recorded, one in 1900 at Gloucester, Massachusetts (Painter), and the other in 1901 at San Francisco and vicinity, California (Woods). In 1905, central Illinois reported 8 cases (Norbury), and in 1906 New York City reported 36 cases (Gibney and Wallace).

In 1907, poliomyelitis appeared for the first time over an extensive area of the United States, with the principal foci located in the Commonwealth of Massachusetts. The origin of the epidemic is in question. Some epidemiologists believe the virus may have been imported by the Scandinavian immigrants who came to this country shortly after the large epidemic in Norway and Sweden in 1905. However, the distribution of the disease did not correspond geographically with the localities in which the immigrants settled. The epidemic began in New York City in June, 1907, and reached its height in September. Exact figures of the number of cases are not available, but they have been estimated at a minimum of 2500 (New York Collective Investigating Committee). Undoubtedly many cases were overlooked because patients often did not apply for treatment until the acute stage was over. The chief sufferers were young children. The disease was mild, the mortality being about 5 per cent.

Lovett and Lucas estimated the cases in Massachusetts at 234. There appeared to have been some evidence of spread of the disease along lines of communication, as the curve of onset was later in the out-of-town cases. It was considered possible that this epidemic represented a spread from the New York focus, for cases were still numerous in Massachusetts in October, although the height of the epidemic had occurred at the same time as in New York City.

Other foci of the epidemic appeared at Oil City, Lehigh, DuBois, and Ridgeway, Pennsylvania (Terriberry and Urey); Galesville, Wisconsin (Jegi); Oceana County, Michigan (Griffin); and Live Oak, Florida (Efird).

The disease continued to prevail throughout 1908, but there were fewer cases than in the previous year. The largest outbreak in New York State was at Schenectady, where 29 cases occurred (Clowe). In Massachusetts there was a decrease in the number of cases, with 136 as against 234 in 1907 (Lovett and Richardson). It was noted that the localities which had been affected in 1907 were comparatively immune.

There was a considerable spread of the disease westward. Minnesota reported 150 cases (Hamilton); Flint, Michigan, 30 cases (Manwaring); Eau Claire County, Wisconsin, 408 cases (Frauenthal and Manning); and Iowa, 9 cases (McCreary). Southward, outbreaks occurred at Clearfield and Lewiston, Pennsylvania (Mills, Barnett); Salem, Virginia (Wiley and Darden); and in Florida (Frost).

After 1908, the disease spread rapidly, gaining in force. In 1909, 923 cases were reported in Massachusetts, and the disease appeared in 136 towns, whereas in 1908 it had been reported in only 47 (Lovett). An analysis of the data collected for the years 1907, 1908, and 1909 in Massachusetts revealed that the disease appeared in scattered foci in all parts of the state, and showed a predilection for river valleys. Koplik[16] reported 1200 cases in New York City and State. He found many cases typical in character and grouped them under the cerebral, neuritic, and classical types. The classical type was found to be the most prevalent.

In 1909, the disease was also epidemic in Minnesota, and the number of attacks was estimated from 800 to 900. In Nebraska, nearly 1000 cases occurred between May and the end of the year. In this state, a regulation was put into effect enforcing a compulsory quarantine of three weeks on all persons who had been exposed to contact. Twelve days after this ruling was made obligatory, the spread was somewhat abruptly arrested.

In 1910 the disease assumed very serious proportions. The United States Census Bureau returns listed 1459 deaths due to infantile paralysis. If 10 per cent is considered the average mortality, the total number of cases would amount to 14,590. The disease was prevalent in forty-three states. In the east, the greatest prevalence was in Massachusetts (845 cases), New York (322 cases), Connecticut (168 cases), and Rhode Island (231 cases). A large epidemic occurred in Pennsylvania, comprising over 1000 cases with a mortality of 22 per cent. Minnesota, Virginia, District of Columbia, Washington, Oregon, and California also suffered large outbreaks of the disease. The largest number of cases in any of the western states was reported from Minnesota, where an epidemic of 1000 cases occurred. In the state of Iowa, 654 cases were reported of which 161 died; 357 cases were reported in Virginia; and 189 cases were reported in Kansas. Other states, including Michigan, Idaho, Ohio, Wisconsin, Vermont, and South Carolina had a few cases.

During the next five years, from 1910 up to 1916, the number of cases in the United States was considerably lower. There were many sporadic in form and widely disseminated throughout the country, but the epidemic foci were smaller. In 1911, the total number of cases registered in the entire United States was only about 2500. The largest outbreaks were in Massachusetts, New York, Pennsylvania, Ohio, Illinois, and Virginia.

By 1912, notification of the disease had been made obligatory in twenty states. The number of cases for that year in the entire United States was nearly 4000, with a mortality-rate of 19.7 per cent. The disease was again prevalent in New York, and 934 cases were reported of which 125 were fatal; 504 of these cases occurred in New York City. Another large focus in New York State was in Buffalo, where over 300 cases occurred, with a mortality of 12 per cent. Massachusetts reported 169 cases, Pennsylvania 267 cases, Ohio 354 cases, Illinois 480 cases, Virginia 229 cases, and California 531 cases.

In 1913 approximately 3000 cases occurred throughout the United States. The largest outbreaks were reported from the states of New York, Massachusetts, Virginia, Illinois, and Indiana. There was also an epidemic of about 50 cases in Texarkana, Arkansas. Sporadic cases were reported from practically every state.

In 1914 there was a considerable decrease in the number of cases; the entire country reported less than 2000. Vermont suffered a large outbreak of about 300 cases that year, with two main epidemic centers, one at Barton, and the other at Burlington. Many abortive cases were recognized in this outbreak. The disease broke out later in the season than usual, and the attack ran late into October. It was noted that the section around Rutland, previously a conspicuous center for poliomyelitis, was nearly free from cases, and Hardwick, which had been attacked the previous year, had none.

So far as may be ascertained, about 2000 cases were recorded during 1915. Ohio reported 466 cases, the largest number of any state. Manifestations were also noted in Massachusetts, New York, Pennsylvania, Minnesota, and Virginia.

1916. THE NEW YORK EPIDEMIC.

Distribution.—In the year 1916, an epidemic unprecedented in geographical extent and severity occurred in the United States. It was distributed over eight states, reaching eastward to Boston, westward to Chicago, and southward to Baltimore. Boston and New York City were the chief centers of the outbreak. Nearly every state in the country suffered; Nevada, New Mexico, Georgia, and California were the only states free from cases.

The epidemic started the second week in June in New York City, along the water front in the Borough of Brooklyn, and extended first toward Bay Ridge and then to the Brownsville section of Brooklyn. The infection spread rapidly along lines of communication, both by railway and water routes, to the towns of Long Island and to New Jersey. By July 15th, the disease was increasing in Philadelphia, Bridgeport, and Camden, and by August 1st it was prevalent in Boston, Baltimore, Chicago. Providence, St. Paul, and Syracuse. A focus of the disease would appear, increase in severity until it reached its height in about six weeks, and then gradually decrease. The epidemic showed its customary irregularity in distribution, in that some places along the lines of public travel, such as Jersey City and Hoboken, located near and in close association with New York City, escaped with only a few cases, while Newark, New Jersey, which was farther away, suffered extremely.

Morbidity. Between June and December of 1916, more than 13,000 cases had been reported in New York State alone, and more than two-thirds of them had occurred in New York City. Doubtless this number did not include many abortive cases, which were not recognized.

A higher proportion of the population was affected in the rural districts than in New York City or in other cities; 1.59 per cent per 1000 population were attacked in New York City, and 2.4 per cent per 1000 in the rural districts of New York State. The highest recorded sick-rate for the year, amounting to a little less than 4 per cent per 1000 population, was in Newark, New Jersey.

Age Incidence.—The majority of cases occurred among children between the ages of one and five years. Of 1984 cases investigated in Brooklyn, 1568 were from one to five years of age, 214 were under one year, and about 200 were over five years.

The age incidence of the cases in the rural districts differed from that of the patients in the city. Outside of New York City, only 55 per cent of the children attacked were under five years of age, and 10 per cent of all persons affected were over fifteen years old; in the city, 90 per cent were under five years of age, and 1.7 per cent of the cases over sixteen years. The contrast is brought out clearly by the mortality-rate. In New York City, 97 per cent of the fatal cases were under fifteen years of age; in the rural districts, 80.9 per cent were under fifteen, and 1 out of every 5 was over fifteen years of age.

Mortality.—The mortality-rate of the epidemic of 1916 was the highest ever recorded. In New York City, it reached 27.2 per cent. The death-rate in Newark, New Jersey, where 1360 cases occurred, was 26.96. The mortality among males in both the city and country was 50 per cent higher than among females.

THE PHILADELPHIA EPIDEMIC.

The value of strict measures of quarantine in checking the incidence of the disease is well illustrated in this epidemic. Philadelphia escaped with a morbidity of only 1 case to 15,000 population. During the entire year, the total number of cases was about 1000. The enforcement of quarantine regulations by the State, a general clean-up of the city, public instruction on the prevention of disease, and the coöperation of civic and health organizations were the measures responsible for the arrest of the disease.

1917–1927.—From 1916 up to January 1, 1927, no large epidemics have occurred throughout the United States. During each year, however, small epidemics and sporadic cases have been reported from practically all parts of the country, the larger incidence being noted, as is customary, in the late summer and fall. In 1917, occasional cases were recorded. It is interesting to note that during that year New York State reported only 70 cases out of 10,000,000 population. The number of cases continued to decrease during the next three years, 2514 having been reported in 1918, 1895 in 1919, and 2291 in 1920. In 1921 the incidence was higher and reached 6229. In 1922, 1923, and 1924 the number again decreased. Approximately 2221 cases were reported in 1922, 3245 in 1923, and 4908 in 1924. During the year 1925, the disease continued to be prevalent throughout the country; 1498 cases were reported for the six weeks ending August 29, 1925, as against 785 cases reported for the same period in 1924.

EPIDEMIC FEATURES.

The wide variation that has been noted in the characteristics of different epidemics and the paucity of statistical evidence make generalization difficult. The features most prominent in all epidemics are the following:

Seasonal Prevalence.—The seasonal relationship has been a factor common to the majority of epidemics. The disease is more prevalent during the warmer months of the year, occurring in July, August, September, and October in the northern hemisphere, and in March and April in the southern hemisphere. There have been exceptions to this adherence to certain seasons, and the disease has shown an epidemic tendency in the winter months. Aycock and Eaton,[17] from a study of the seasonal variation in the morbidity-mortality ratio of the disease over a period of several years in the United States, concluded that infantile paralysis prevailed to a greater extent during the months from December to May than is commonly reported. They also noted a definite increase in its occurrence in March or April. From a computation of the morbidity and mortality reported by months in the registration area of the United States for 1921, the case fatality-rate was found to be much higher in the winter and spring than in the summer and autumn, thus reaching its highest point at the time of least prevalence, and dropping to its lowest at the season when the disease prevails to a great extent. In the opinion of Aycock and Eaton, this variation is due chiefly to the failure of physicians to recognize the mild type of the disease in the season when poliomyelitis is ordinarily quiescent, as well as to the fact that deaths are reported more promptly than cases. If the seasonal morbidity is calculated upon the more accurate mortality reports, then the number of cases in the season of least prevalence, that is, from December to June, is considerably larger than the incidence reported for these months.

Many writers have tried to show a relation between a dry season and the occurrence of the disease, but such a dependence is not confirmed by experience. A study of the rainfall shows that epidemics have occurred during a wet season as well as when there was little rain.

Periodicity. The tendency for epidemics to recur has never been satisfactorily explained. Flexner[1] has shown experimentally that the virus tends to increase in activity until it reaches a high point. It maintains this state of virulence for a long period, even up to three years, and then suddenly becomes avirulent. Possibly several factors are responsible for the recrudescence of the disease after a lapse of years. The virus may be imported from a locality which has suffered a recent epidemic. It may survive in a latent state from a previous epidemic and suddenly become active. On the other hand, the relatively low susceptibility of individuals suggests that many acquire immunity by exposure in other epidemics, so that the disease is not likely to recur until there is a fertile nucleus.

Susceptibility.—There seems to exist a wide variation in individual susceptibility, even within one family. Children openly exposed to the disease, even occupying the same bed with the sick child, have not contracted infantile paralysis. Susceptibility is present in only about 2 per cent of the children under thirteen years of age, which rate is decidedly less than that of the predisposition to other communicable diseases of childhood. Whether there is any system of natural defense against the invasion of the infection, which may help to account for this individual susceptibility, is as yet a matter of conjecture. Amoss and Taylor,[18] in a series of 56 experiments, demonstrated that the washings of nasal and pharyngeal mucosæ of apparently healthy persons possess a definite power of neutralization or inhibition. This power was found to be a variable property, as washings taken at different times from the same person neutralized the virus in some experiments, but had no effect in others. Sufficient tests have not yet been made to determine to what extent this neutralizing or inactivating power may act as an element of defense.

The possibility of establishing individual susceptibility by means of a serum test may be worthy of consideration. Such an examination would have the same value as the Schick test, which determines the presence of immunity to diphtheria. Of course, there would be several difficulties encountered in making such a test in poliomyelitis. The fact that the actual bacterial cause of infantile paralysis has not yet been definitely established would make such a procedure difficult. It would be necessary to determine whether serum from recent cases, or substances from the secretions of the nose and throat of active cases, were better for testing purposes. A great obstacle would be the possibility of a mixed infection. But in spite of these apparent difficulties, it seems feasible that some such test might be worked out and made on children of the most susceptible age.

Children between the ages of two and five years are the most susceptible. The period of greatest susceptibility is the second year of life. Infants are rarely affected, possibly because they are seldom in contact with other children. After the fifth year, the incidence declines. In the country, the age of those affected is higher than that of cases in the city. This difference was brought out in the study of 4186 cases made by Shaw[19] in the 1916 epidemic in New York

State; 90 per cent of the cases in New York City were under five years of age, but in the rural districts only 55 per cent were under five and 10 per cent were over fifteen. It is possible that the adults in the city developed immunity by being exposed more frequently to the disease.

It is difficult to explain the susceptibility of children between the ages of two and five years. The theory advanced by some investigators is that poliomyelitis, with its pathological affinity for ganglionic motor cells, finds ready victims among children between the ages of two and five years—an acute developmental period -during which activity renders the spinal areas vascular, thus increasing the susceptibility to infection.

In general, the susceptibility of boys and girls seems about equal, although there has been evidence in nearly all epidemics showing that the proportion of boys attacked was slightly higher. This may be due to the fact that a boy, being active, is more exposed to the infection.

No particular racial susceptibility to the disease has been evident, although in some epidemics certain races seem to have shown a somewhat lower incidence.

In regard to the type of child who seems more susceptible, Draper[20] mentions the large, plump, apparently sound child. The fact that illness in healthy children attracts attention more than disease in children of weaker constitution may account for this observation. As to the adolescents attacked, Draper says that their type is usually opposite to that of the child, in that they are of delicate physique. Various anatomical irregularities have been mentioned by different writers, but it is difficult to find any causal relation between them and the disease.

Immunity.—It is the general opinion that one attack of poliomyelitis immunizes a person against subsequent attacks. Flexner and Lewis[21] have proved experimentally that a monkey that has had the disease is immune to further inoculation. Communities where poliomyelitis has occurred seem to acquire a certain degree of immunity, as they escape entirely or are only slightly affected in a subsequent outbreak.

Rare instances of relapse or recurrence of the disease have been reported. Francis and Moncreiff[22] collected 14 such cases from literature. Usually the recurrence took place within a few days or weeks of the original attack. Other cases of reinfection with longer intervals between attacks have been reported. Lucas and Osgood[23] recorded one case of recurrence more than two years after the initial attack; Taylor[24] reported one that occurred three years after the first illness; Eckert[25] one after a six years' interval; and Sheppard[26] one after sixteen years.

The cause of these secondary infections has not yet been determined. In the case of the occurrence of a second attack after a short interval, it would seem that the original infection flared up again. That the virus may be present in the nasal secretions for a long time after the attack has been demonstrated by Lucas and Osgood.[23] The Royal Medical Institute of Sweden[27] reported that experiments during the epidemic of 1911 demonstrated the persistence of the infection in the mouth and intestinal tract for long periods, varying from two weeks to seven months after the onset of the disease. In the case of longer intervals between attacks, the child evidently did not develop immunity, or immunity became exhausted, leaving the patient subject to an entirely new infection. Just how long immunity lasts has not been determined.

Incidence.—Due to the general low susceptibility of individuals to the full pathogenic power of the poliomyelitic virus, the incidence in the total population is small. In the cities the morbidity-rate in epidemics is seldom greater than 1 per 1000 population. Instances of larger case-rates have been reported from small towns and rural districts. In the epidemic of New York City in 1916, the case-rate in the city proper was 1.59 per cent per 1000 population, and in the rural districts 2.4 per cent per 1000 (New York Monograph[6]).

Social conditions and environment seem to have had little bearing on the incidence. The disease appeared in the better class of dwellings as well as in the thickly tenemented districts. In spite of no apparent variation in the incidence among different social and economic conditions, the spread of the disease undoubtedly is affected by a lack of observation of hygienic measures, and crowded living conditions.

Mortality.—There has been a marked fluctuation, ranging from 5 per cent to 40 per cent, in the mortality of the various epidemics. Aycock and Eaton,[17] from a study of the mortality-rate in thirty-eight epidemics which occurred from 1894 to 1921 in various countries, estimated the average mortality-rate at 20.8 per cent.

As to the ages at which the disease is most likely to be fatal, Nicoll,[28] in a study of fatalities in the New York State epidemic of 1916, found that the disease caused the greatest number of fatalities among infants under one year of age and among persons over fifteen. After the third year, the fatality tended to increase with age. It seemed that up to ten years of life, the country child had a better chance of surviving than the city child, but that adults had the advantage in the city.

Modes of Transmission. – There are many theories as to the manner in which the disease is spread. The upper respiratory tract has been pointed out by Flexner and Lewis[29] as the means of ingress of the infection into the body. Demonstration has shown that the virus may reach the subarachnoid space through the lymphatics of the nasal mucosa. But the prevalence of the disease in the summer months, when respiratory diseases are at their lowest point, cannot be explained on this basis. The exact method of the transmission of the infection is not yet well established, but it is probably propagated through secretions by way of Flugge's droplet infection, carried to the upper respiratory mucous membrane either by direct or indirect personal contact, or by mechanical transference of the virus from infectious secretions and dejecta.

Human Carriers.—There seems to be little doubt that human carriers play a part in the diffusion of poliomyelitis, since the virus has been proved to exist upon the mucous surfaces of the nose and throat. The infection may be conveyed by persons suffering from an acute typical attack of the disease; by those having a mild or atypical form; by healthy persons who have been in contact with the sick, but who themselves have not developed the disease; and by chronic carriers who have apparently recovered from a typical attack (Flexner[30]). It is impossible, however, to estimate even approximately the number or distribution of such carriers.

Wickman[4] and Flexner[30] are the chief advocates of the theory that the disease is communicated by direct contact in sneezing, coughing, kissing, and even loud speaking. Such a conclusion is based on experimental and clinical observations. Wickman[4] cited cases of infection, with a village school as a focus, in the epidemic of Trastena. In the 1914 epidemic in Vermont, 68 out of 304 cases were found to have been in contact with either frank or abortive cases.[31] Nicoll,[32] in studying 756 cases in the New York City epidemic of 1916, found that contact was an immediate factor in the spread of 20.4 per cent of the cases. Numerous other instances may be cited in which there were evidences of spread through contact.

Some inconsistencies are found in this theory. Definite contact can be traced in only a small proportion of cases. The occurrence of more than one case in a family is uncommon, and where multiple cases do occur in a family, it is questionable which ones are truly secondary. Aycock and Eaton,[33] from a study of a series of multiple paralyzed cases in 253 families, concluded that a large proportion of the secondary cases can be traced to the same source as the primary cases. The true secondary cases are probably those that occur on an average of fourteen days after the first case, and the proportion of these is much smaller than those cases that apparently have a common source. In the epidemic of 1916, only 4 per cent of 8635 families attacked suffered more than one case (New York Monograph[6]). In the Pennsylvania epidemic of 1910, there were 289 instances of well children sleeping in the same room with paralyzed cases, but only 24 contracted the disease (New York Monograph[6]). It is evident from the above inconsistencies that other factors must play a role in the spread of the disease.

Mechanical Transference.—The theory of the mechanical transference of the infection is well supported by experimental and clinical evidence. Since the virus is not affected by drying, it is possible that it may be present in dried excreta, which is disseminated in several ways.

Dust.—It may become carried in the form of dust. Thro and Neustaedter,[34] and Josefson[35] have proved experimentally that the virus exists in the dust of the sick-room. Müller[36] is a believer in the theory of the transmission of the virus by infected dirt on clothes or other objects. He noted that in the epidemic of Hesse-Nassau the disease first developed in families whose bread-earners were in contact with the outside world, such as the shoemaker and the inn-keeper.

Foodstuffs.—There have been instances pointing to foodstuffs as carriers of the infection. Dingman[37] found milk to be the medium by which the disease was spread in 7 cases. A boy at a dairy contracted poliomyelitis. Seven customers, who used the milk from this dairy unboiled, developed cases in their families, but those who boiled the milk escaped. Aycock[38] also traced the outbreak of a small epidemic to contamination of a milk supply. A boy who worked in a dairy contracted poliomyelitis. This dairy supplied two routes with milk. Cases developed in all the houses on the first route and 1 case occurred at a house on the second route, where a few bottles of milk left over from the supply allotted to the first route were delivered.

There also seemed to be some evidence of milk as a carrier in the 1909 epidemic in Massachusetts, in which none of the children living on breast-milk alone were affected, although 70 per cent of the cases had been in the first year of life (Lovett[39]). There have been instances, however, of breast-fed children having the disease, so milk is not the only source of transmission.

Foods that have been exposed or kept too long in ice-chests may play a part in the dissemination of the virus. The fact that epidemics occur in warm weather, when fruits are exposed and when food spoils easily, points to foodstuffs as agents.

Insects.—In view of the fact that the curve of insect life coincides closely with the outbreaks of poliomyelitis, which occur chiefly in the warm months, several kinds of insects, including flies, mosquitoes, and fleas, have been suspected of acting as carriers. It is easily conceivable that insects may feed on the excreta of the sick and track the infectious virus wherever they light. Such a possibility has been demonstrated experimentally. Flexner and Clark[40] and others have found that a fly can carry the virus in a living state for forty-eight hours or more.

The possibility of the insect's incorporating the infection into its organism, and transmitting it in biting, is supported by only isolated experiments and by little clinical evidence.

Domestic Animals and Poultry.—Several other theories have been advanced to explain the dissemination of the virus. From time to time animals and poultry have been considered as possible carriers, due to the not infrequent occurrence of paralysis among fowl and domestic animals. There are objections to this theory, in that the disease among animals does not always show the characteristics of human poliomyelitis, and experimenters have not been successful in transmitting a paralytic disease from one animal to another.

Richardson[41] advanced the theory that infantile paralysis is always present among rats, but appears among humans only when the rat infection and the flea infestation are at their maximum. Although some observers have seen rats with paralyzed legs in a locality where children had poliomyelitis, in general there has been no epidemic disease noted among rats at the time when outbreaks occurred among humans. Laboratory investigations are also needed to substantiate the assumption that rats are active carriers.

It is possible that mice may act as passive agents by coming in contact with infective secretions.

Several epidemiologists have noted the occurrence of paralytic diseases among domestic animals coincidentally with epidemic poliomyelitis among humans. Paralyses among horses, dogs, and cats have been observed most frequently. Flexner[42] discredits the hypothesis that animals are active agents, as it has been found practically impossible to infect domestic animals except by inoculation, and a sick animal has not transmitted the disease to a healthy animal.

The prevalence of a paralytic disease among poultry has been observed very frequently during epidemics of human poliomyelitis. Caverly[31] noted instances of paralytic affections among hens in practically all the epidemics in the state of Vermont. Flexner,[43] however, found no etiological relation between the lesions of the paralytic disease among fowl and those of human infantile paralysis; on the contrary, the affection from which poultry suffered seemed to be due to lesions of the peripheral nerves.

Haynes[44] believes the solution of the relationship of animals and insects to the occurrence of an epidemic is delayed by our lack of a standard for recognition of manifestations of poliomyelitis among the lower animals; that is, the infectious organisms of poliomyelitis may produce lesions and signs among the animals other than those recognized in man, and in this way the contagion may be kept alive. The higher life is affected only at times.

Miscellaneous Theories.—The virus has also been considered air-borne, water-borne, or as originating in damp earth. Such theories have not been based on sufficient evidence to be of value.

PROPHYLAXIS.

General Precautionary Measures.—In view of our lack of definite knowledge as to the means of transmission of the disease, we cannot deduce concrete prophylactic rules, but must provide sufficient measures to cover every possibility. During the epidemic of 1916 in New York City, the Department of Health issued many pamphlets in which were listed regulations to be followed in times of epidemic. The measures advised in the following pages are based upon these reports.

1. Do not let the child travel beyond ordinary rounds. Home life favors a preventive program. Isolation of children, carried out even in the midst of districts where the disease is prevalent, may suffice to protect the child from infection, regardless of the fact that he is using the same water and food supply. The fact that the disease did not develop in forty-five charitable institutions, which maintained a rigid quarantine during an epidemic, demonstrates the value of isolation.

2. Avoid all food and drink except at home. Eat only freshly cooked food, and sterilize both milk and water. Avoid all raw fruit that has not been washed.

3. Do not handle pets.

4. Protect the child, and also food, from all insects and vermin. Keep all food, likely to spoil, on ice.

5. Keep the child from kissing, fondling, and handshaking.
6. Avoid bathing and swimming in polluted water.
7. Have the least digestive disturbance attended to.
8. Promote general cleanliness by keeping the house clean, by screening windows against flies, by destroying refuse, and by keeping the body and clothing clean.

The value of the use of antiseptic solutions in the nose and throat is questionable. Amoss,[45] in a small series of experiments, proved that immune bodies were present in the nasal mucosa. It is probable that a large majority of individuals possess some such such means of protection. Any solution introduced into the nose might destroy the neutralizing substances. Furthermore, it has been shown experimentally that antiseptic solutions used in the nasal cavities of monkeys do not prevent the disease.

Precautionary Measures in Case of Exposure.—In case the child has been definitely exposed to the disease, it will do not harm to use hexamethylenamin. Flexner and Clark[40] found that if monkeys were given hexamethylenamin before an intracerebral injection of the virus and daily administrations by mouth after the injection, the incubation period was prolonged in some animals, and the onset of paralysis entirely prevented. In the case of children, 1 to 5 grains four or five times a day should be administered according to age. The use of the drug should be stopped on the appearance of any urinary irritation.

In regard to the isolation of those who have been in contact with the disease, the regulation of the Health Department of New York City provided that the quarantine period of children who had been, but no longer were, exposed to infection, should be limited to fourteen days. Children under sixteen years of age, who did not have the disease, but who remained at home in families in which poliomyelitis had occurred, should be isolated in the home until two weeks after the termination of the case by death, removal, or recovery. Persons over sixteen years of age in families where there were cases of poliomyelitis might continue working, provided their business did not involve the preparation and handling of food, and if they were not in contact with children under sixteen years of age. Adults and children under sixteen years of age, affected by this regulation, might resume their occupations after two weeks if they removed to a different place of residence.

Precautionary Measures in Case of Infection.—It is generally agreed that isolation of the patient for two or three weeks from the date of onset is advisable. This period seems adequate, as there are many cases in which the diagnosis is not made until late, and yet no secondary cases develop.

If the person is not removed to the hospital, it is very important to observe the following regulations:

1. The patient should have a separate room. Only the attending physician and nurse should be allowed to enter it. The room should be well-aired, clean, and the windows screened.

2. The attendant must not perform any household duties for other members of the family. She may leave the house, provided she takes the necessary precautions as to personal cleanliness, and avoids all children.

3. All discharges from the nose and throat, and excreta should be disinfected. All eating utensils, personal clothing, and bed linen should be disinfected.

4. The nurse should wear a cap and gown of washable material, over her ordinary clothes. She should wear a gauze nose and mouth protector. The physician should also wear a gown and mask. There should be as little contact with the patient as possible. Before leaving the patient, the nurse and physician should scrub their hands thoroughly with soap and hot water.

Placards.—All premises where a case of poliomyelitis occurs should be placarded (the only exceptions being hotels and boarding houses, provided the patient is at once removed to a hospital, the room or rooms immediately disinfected, and no quarantined children allowed to remain on the premises). In private houses and in two-family houses with separate entrances, a placard should be placed on the door leading to the room the patient occupies. In apartment and tenement houses, a placard should be posted on the door of the apartment occupied by the family of the patient. In two-family houses with a common entrance, one placard should be placed on the door entering the portion occupied by the family of the patient, and one upon the room or rooms occupied by the patient.

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CHAPTER III.

ETIOLOGY.

Historical.—Poliomyelitis was established clinically as an infectious disease by Strümpell,[1] Medin,[2] and Wickman.[3] In 1909, Landsteiner and Popper[4] experimentally corroborated this clinical evidence, when they succeeded in transmitting the disease from man to monkey by an intraperitoneal injection of a small quantity of an emulsion of the cord of a child who had died of poliomyelitis. This experimental transmission of the infection to monkeys was soon confirmed by other investigators, notably Landsteiner and Levaditi[5] in France, Flexner and Lewis[6] in America, and Leiner and von Wiesner,[7] Römer,[8] and Krause and Meinicke[9] in Germany. Flexner and Lewis were the first to use the intracerebral route of inoculation and to transmit the disease from monkey to monkey. The establishment of a method, by which the infection might be almost constantly produced, afforded opportunity to study the pathogenic agent as to its nature, its method of ingress and egress, its distribution throughout the body parts, and its powers of immunization.

The search for the specific organism causing the disease was diligently pursued. The divergence of the results of the different investigators was remarkable. The staphylococcus, streptococcus, pneumococcus, and numerous other bacteria were suggested as the etiological agents. The Gram-positive diplococcus and tetracoccus, isolated from the spinal fluid by Geirsvold,[10] received most attention, but attempts to cultivate these organisms and to produce the disease by injection of them were unsuccessful. Dixon, Fox, and Rucker[11] suggested, as a cause, a bacillus that they found in the blood of 10 human cases of poliomyelitis, but it was not possible to demonstrate this agent on stained smears of the cord or brain. No two investigators found the same pathogenic agent, and none of the bacteria suggested, when injected into monkeys, produced pathological lesions characteristic of poliomyelitis.

Further bacteriological research was discouraged by the discovery that the virus was filterable.[5, 8, 9, 12, 13] In 1913, Flexner and Noguchi[14] announced that they had succeeded in isolating an organism from the central nervous tissue of cases of poliomyelitis, to which they gave the name "globoid bodies." More recently, Rosenow and his co-workers[15] have made studies of the cultivation of a streptococcus which they obtained from the central nervous system and from the tonsils of human and monkey cases. Further comment will be made on the studies of Flexner and Noguchi and on those of Rosenow and his associates.

THE VIRUS.

The Nature of the Virus.—The virus belongs to the filterable class, as it will pass through the pores of a Berkefeld filter and asbestos. It withstands exposure to light, drying, freezing, heat, and glycerination for a long time. Even at the height of summer heat and when dried on clothing, it retains its virulence. Ordinary weak chemicals, such as those contained in the solutions commonly used in the nose and throat, have no effect upon the virus and therefore do not aid in preventing the development of the disease. Five-tenths per cent solution of phenol, applied for three days, does not render the virus inactive; bile has no effect upon it. Its activity persists in water and milk after thirty days. It may be frozen at −2° C. to −4° C. for four days, without showing material damage.

Conditions which lead to the destruction of the virus are exposure to heat 50° C. for one-half hour, exposure to the sunlight, and the action of 0.5 to 2 per cent solutions of hydrogen peroxide, menthol solutions, iodine, and other stronger disinfectants (Ruhräh and Mayer[16]).

The virus shows fluctuation in virulence in both the human body and in monkeys. In man, the virus is probably most infective during the first two weeks of the disease, after which time it begins to be attenuated. It may, however, retain its strength over long periods. Flexner and Amoss[17] infected monkeys by injection of nervous tissue, which had been taken from human beings and monkeys suffering from poliomyelitis, and preserved in 50 per cent glycerol at refrigerator temperature for a period of four to six years. The symptoms and lesions produced were identical with those caused by injection of the virus obtained from fresh material of the spinal cord and medulla. In the monkey, the virus possesses a comparatively weak pathogenic action. With successive passages, its power tends to increase and to become fixed, until finally the virulence is so great that only a small quantity produces infection. This intensity endures for some time, but finally diminishes (Flexner[18]).

The fluctuation in the infective power of the virus corresponds to the changes in virulence of the disease in times of epidemic, when the number of cases seems to increase, persist in intensity for some time, and then decrease. Flexner considers these fluctuations in virulence to be dependent upon causes acting upon the virus from within the body of the monkey.

The Method of Ingress and Egress of the Virus.—In the experimental study of poliomyelitis, the mucous membrane of the nasopharynx has been found involved to such an extent that the upper respiratory tract is generally accepted today as the portal of entry and exit of the disease. It has been established that the virus is present in the mucous membranes and secretions of the nasopharynx of the ill, and in washings from the nasopharynx of those who have been in contact with the disease. In experimental poliomyelitis, Flexner and Lewis[19] detected the virus in the nasopharyngeal mucous membrane by injection of a filtrate prepared from the excised nasal mucous membrane of infected monkeys. Landsteiner, Levaditi, and Pastia[20] first detected the presence of the virus in the pharyngeal mucosa of a case of human poliomyelitis.

The virus was detected in the nasal secretions of paralyzed monkeys by Landsteiner, Levaditi, and Danulesco,[21] who inserted cotton plugs into the nares of the animals and infected the well monkeys with the absorbed solution. The first demonstration of the virus in the nasopharyngeal washings of healthy persons was made by Flexner, Clark, and Fraser.[22] The nasal cavities of the parents of a child suffering from poliomyelitis, neither of whom showed any symptoms of illness, were rinsed and the filtrate injected into two monkeys. Typical poliomyelitis was produced. Kling and Pettersson[23] also experimentally demonstrated the existence of the active virus in the nasopharynx of the healthy members of a family, one of whom had died of acute poliomyelitis. In the observations by Flexner, Clark, and Fraser and in those by Kling and Pettersson, mixed washings were used, so that it was not possible to recognize individual carriers. Taylor and Amoss,[24] by irrigating the suspected carriers separately, found that more than one member of a family may harbor the organism. They also cited a case showing that a healthy carrier may develop poliomyelitis.

The ease with which the monkey may be infected by the injection of the virus into the nasal cavities points to this route as the entrance of the disease. Landsteiner and Levaditi[25] infected monkeys by direct inoculation of the virus into the nasal mucosa. Leiner and von Wiesner,[7] by simply painting the nasal mucosa with a highly adapted virus, produced experimental poliomyelitis.

By what route the virus enters into the nervous system from the nasopharyngeal mucosa has not been definitely determined. It may penetrate into the bloodvessels and be carried into the nervous tissues by the circulation, or it may penetrate into the nervous tissue by way of the sheaths of the olfactory nerve filaments. Experimental demonstrations have supported the latter route. When the virus has passed into the lymphatic vessels surrounding the olfactory nerve, it penetrates into the subarachnoid space and is disseminated by means of the cerebrospinal fluid, following the invaginations of the arachnoid along the spaces of the anterior spinal vessels into the interior of the cord. Such a path may be traced by the advancing columns of cells which are thrown out in reaction all along the course of the vessels to their terminations about the anterior horn cells (Draper[26]).

The difficulty of infecting monkeys by intravenous injection of the virus strongly suggests that penetration of the virus into the nervous tissue is by way of the olfactory nerve filaments. Flexner and Amoss[27] found that when the virus was injected directly into the blood, there was tendency for it to be withdrawn from circulation into the bone-marrow and spleen instead of into the nervous system. The function of the choroid plexus and the pial lymphatic vessels plays an important part in excluding the virus from the nervous system. Flexner and Amoss[28] found that any disturbance of the choroid plexus mechanism, as for instance a sterile meningitis, removed its protective power and permitted the virus to penetrate into the nervous system.

Draper[26] has sought to explain the different types of cases on the the basis of the circulation of the virus in the blood, as follows: In the abortive type, the virus apparently never reaches the nervous system; in the type called "dromedary" by Draper, the virus, while in the blood produces general symptoms and later, when it has gained entrance into the nervous system, results in paralysis; in the type in which the symptoms of the general infection and the nervous disturbances are continuous, the virus is probably more virulent, or the patient has less resistance. There is still another group of cases in which the nervous symptoms are manifest at the beginning of the disease, due to the virus reaching the nervous system immediately upon its entrance into the body.

The nasopharynx has been suggested also as the portal of exit of the virus. Experiments by Amoss and Taylor[29] have shown that when the virus is introduced into the central nervous system, evidences of its presence appear in the mucous membranes of the nose and throat.

The Distribution of the Virus in the Human Body.—The presence of the virus in the various body tissues must be determined by inoculation, since the virus is not visible to the eye. Tissues obtained from autopsic materials of human beings, body fluids from living cases, and human tonsils and adenoids removed surgically during life have been used in experimental work. The virus has been demonstrated in the brain, cord, medulla, basal ganglia, cervical and lumbar enlargement, posterior root ganglia and their cranial analogies, and in the sympathetic ganglia. In the organs outside the nervous system, it has been found in the nasal and pharyngeal mucosæ, the tonsils, the mesenteric glands, and lymph glands (Amoss[30]).

The tonsil is considered by some investigators to play an important role in the disease. Flexner and Clark[31] reported successful inoculations with tonsillar and pharyngeal mucosæ. They found that the ordinary filtrate from these tissues failed to cause the disease, but when the material was rendered free of bacteria and inoculated as suspensions, infection was produced.

The failure to demonstrate the presence of the virus in the human spinal fluid causes some wonderment. Abramson[32] experimented carefully along this line, but did not detect the virus in any of his observations. In one experiment, cultures made from more than 1200 fluids taken from cases in all stages of the disease were sterile, except in a few cases of contamination. In another experiment, fluids were collected from 40 patients, centrifuged at high speed for forty-five minutes, and the sediments injected intracerebrally into monkeys. No effect was noted.

In certain experiments on monkeys the virus has been found to be present in the spinal fluid. Flexner and Lewis[33] demonstrated that after a considerable dose of filtrate was injected, the fluid withdrawn on the third day before paralysis set in contained the virus, and injection of the fluid intracerebrally into another monkey produced the disease.

The presence of the virus in the human blood has not yet been demonstrated. As a rule, inoculations with specimens of monkey blood have not produced the infection. In only 1 experiment out of 10 were Clark, Fraser, and Amoss[34] able to produce the disease by an intracerebral inoculation of a specimen of blood removed at the onset of the paralysis. These same experimenters found that the blood of monkeys, which had received intracerebral injections or intracerebral and intraspinal injections of suspensions of the spinal cord of a paralyzed monkey, when removed from one to forty-eight hours after injection, failed to produce the infection when inoculated intracerebrally into another monkey. Filtrates injected in small amounts into the blood usually failed to cause the disease, and although large amounts caused the blood to remain virulent for seventy-two hours, the infectiveness disappeared by the eleventh day. Apparently some protective mechanism excludes the virus from the blood.

The Persistence of the Virus in the Body Parts.—How long the virus persists in the body tissues and fluids has not yet been determined, because of the difficulties in carrying out experimental work in poliomyelitis. In monkeys, the virus has been found to disappear from the central nervous system in three to six weeks. In man, the virus grows rapidly weaker after the first eight to fourteen days and usually has disappeared entirely by the end of the fourth week (Ruhräh and Mayer[16]).

The virus is usually present in the nasopharyngeal mucosa during the first week, especially in fatal cases. It tends to disappear as the disease advances, and is rarely found after the tenth day (Flexner and Amoss[35]). Lucas and Osgood[36] reported a few exceptional cases in which the virus was retained in the nasopharyngeal tissue at the expiration of six months. In nasal washings the virus is generally found greatly diminished in amount after the first week, but, here again, there are exceptions. Lucas and Osgood reported the presence of the virus in nasal secretions four months after a second attack. It would seem that the period of greatest infectivity is early in the disease.

Defenses of the Body Against the Invasion of the Virus.—The infectious agent must encounter considerable resistance, as only a small percentage of the population is affected, and there is a tendency for only one member of a family to suffer a true attack of the disease. Experimental work has revealed that neutralizing or inactivating substances are present in the secretions of the nasal and pharyngeal mucosæ of healthy persons. Amoss and Taylor,[29] from a study of 56 experiments, showed that nasal and pharyngeal washings exercise a neutralizing reaction on the virus. Normal anatomical conditions were present in the nasal and pharyngeal mucosæ of the healthy persons whose nasal secretions were used in experiments. The neutralizing or inhibiting power was not constant, but varied in a given individual. The presence of inflammatory conditions in the upper respiratory passages tended, in most cases, to lessen or entirely destroy the power. Whether children and adults differ with respect to this neutralizing power has not yet been determined.

The nasal washings of those suffering from poliomyelitis may or may not exercise inhibitory power on the virus.

The neutralizing substances are water-soluble and appear to be organic. They are more or less thermolabile.

Another body defense against the invasion of the virus is the meningeal choroid plexus. Flexner and Amoss[28] have experimented widely to determine the relation between the meningeal choroid plexus and poliomyelitic infection. They found that the choroid plexus mechanism normally excluded the virus from the cerebrospinal tract, whether the infection was promoted through the intranasal or the intravenous route. Any disturbance of the choroid plexus, as by the injection into the meninges of any fluids such as horse serum, normal monkey serum, Ringer's solution, or Locke's solution, served to diminish or remove the protective function, and permitted the virus, introduced intravenously, to pass into the nervous system. Flexner and Amoss also found that the disease could be produced by intranasal injection of the virus, provided an intraspinal injection of horse serum had been given previously. From these experiments, it may be concluded that any slight change in the structure or function of the meningeal choroid plexus may be sufficient to break down an individual's protective power, and afford an opportunity for the virus to penetrate into the nervous system. Of the many substances that have been injected to test their effect upon the meningeal choroid plexus structure, immune serum alone has not set up a disturbance, and no infection has resulted from the subsequent intravenous injection of the virus. Lumbar puncture, if not accompanied by hemorrhage, does not affect the entrance of the virus into the nervous system, but in case of any hemorrhage, permeability is increased.

Whether these defenses are the only sources of protection, or whether they are the essential ones, may be only conjectured. There may be other protective powers present, which have not yet been ascertained.

Immunization.—In human beings, one attack of poliomyelitis ordinarily ensures complete immunity. It is only in exceptional cases that reinfection occurs. In experimental poliomyelitis, Flexner and Lewis[37] have demonstrated immunity to reinfection in several recovered monkeys which failed to contract the disease when reinoculated, whereas paralysis resulted in the control monkeys.

Attempts have been made to confer immunity by preventive inoculation, but experimenters have met with little success. No means have yet been discovered by which the virus can be attenuated artificially. Abramson[32] experimented with the monkey virus attenuated by heat. None of the 11 animals treated showed any ill effects, and 5, out of 8 of those subjected to an intracerebral lethal dose of the virus, resisted successfully. The sera of all the animals were tested and found to contain neutralizing substances in such concentration as should be able to combat a comparatively mild infection. Other experimenters have not obtained success with this method of attenuation.

Flexner and Amoss[38] have sought to acquire a strain of the virus that would be only mildly effective in the monkey. Recently, they have encountered such a strain; it produces a paralysis in monkeys, but instead of the infection ending fatally, it tends to terminate in recovery. In controlled experiments, this mild infection has been found to leave the monkeys protected against inoculation of a virulent strain of the virus. This strain was discovered only recently, but during the two years in which it has been studied, its potency has been found to remain fixed.

The immunization afforded by an attack suggested the possibility of the blood's containing neutralizing substances. Netter and Levaditi[39] found that the serum of a convalescent patient would neutralize the virus *in vitro.* Römer and Joseph,[40] and Flexner and Lewis[19] found that the serum of recovered monkeys and of human beings who had suffered either an abortive or true paralytic attack would protect normal animals against fatal doses of the virus. Flexner and Amoss[39] showed that these neutralizing substances appeared in the blood by the sixth day of the disease.

The application of serum treatment in human cases was first made by Netter and his co-workers.[41] Many investigators have tried to determine its value, but at present there is a variance of opinion as to its efficacy. A detailed discussion of the use of serum will be found in the Chapter on Serum Treatment.

THE ORGANISM CAUSING POLIOMYELITIS.

In 1913, Flexner and Noguchi[14] announced that they had isolated an organism of peculiar type from the spinal cord of monkeys and human beings suffering from poliomyelitis, to which they gave the name "globoid bodies." Further study revealed that these bodies are constantly present in the central nervous system of both humans and monkeys suffering from poliomyelitis. They are not found associated with any other disease. These organisms have been obtained in pure cultures; inoculation of them into monkeys has produced typical poliomyelitis; and in turn, the organism has been obtained in pure cultures from infected animals. Thus, Koch's requirements have been fulfilled. It has also been found possible to render the organism innocuous by treatment with serum from recovered cases of poliomyelitis.

Amoss[42] has reported the cultivation of the same microörganism from the blood of infected monkeys.

Cultivation of the Organism.—Flexner and Noguchi[14] have succeeded in cultivating the organism by the use of the method utilized for growing spirochetes. Human ascitic fluid, to which has been added a fragment of sterile fresh tissue, is used as the culture medium. The material used for cultivation is nervous tissue, taken preferably from the brain of cases of human poliomyelitis and infected monkeys. If contamination is suspected, filtrates are made from the nervous tissue. As strictly anaërobic conditions are essential to the growth of the initial cultures, the tubes used for the tests are covered with a deep layer of sterile paraffin oil. Growth of the cultures is slow and shows best about the sixth or seventh day.

In size, the organisms are described as very small and globular, measuring from 0.15 to 0.3 micron in diameter. They are arranged in pairs, short chains, or small masses, according to the conditions of growth and multiplication. They resist freezing, phenolization, and glycerolation. They are devoid of independent motility. The organisms may be stained either by Giemsa's method or by Gram's method.

The observations of Flexner and Noguchi have not yet been largely corroborated by other investigators. Their work, in so far as it has gone, is based on authentic principles, and is very important experimental data. Further investigation in the attempt to identify a germ that may be cultivated by all investigators, that will produce typical poliomyelitis, and that may be demonstrated on stained sections, will continue to form one of the most important fields of research.

The Cultivation of the Micrococcus by Rosenow and Co-workers.—Among the theories advanced as to the cause of the disease is the claim that certain strains of streptococci are the etiological organisms. Several independent investigations dealing with the cultivation of a micrococcus, isolated from various tissues of cases of poliomyelitis, have been recorded. Mathers[15] was the first to publish a report on the discovery of this organism. In 6 out of 7 experiments, in which the brain and cords of fatal cases were used for material, Mathers obtained a pure culture of a Gram-positive micrococcus. The organism was also demonstrated in the mesenteric lymph nodes. It was not found in the cerebrospinal fluid or in the heart blood. Rosenow, Towne, and Wheeler[15] cultivated from the brain and cord in 12 cases, and from the tonsils in a large series of cases, a polymorphous streptococcus which was apparently the same organism that Mathers discovered. These investigators also obtained the organism from the ventricular fluid after death, and from the blood in one case, but they did not find it in the spinal fluid. Nuzum and Herzog[15] obtained the same organism from the brain, tonsils, and mesenteric glands, and in addition isolated it from the cerebrospinal fluid obtained by lumbar puncture during life.

The discoverers of this organism claim it to be polymorphous, with the tendency, when grown under anaërobic conditions, to become very small. It its minute stage, it resembles the globoid bodies described by Flexner and Noguchi, and will pass through a filter. Rosenow concluded that the globoid bodies were of the form that the polymorphous streptococcus assumed under anaërobic conditions.

Injections of the cultures of this streptococcic organism into young rabbits, guinea-pigs, and monkeys produced lesions of the central nervous system.

Certain objections have been raised to the theory of a causal relationship between this streptococcus and poliomyelitis. In the first place, the lesions produced by injection of the cultures have showed some departure from the typical symptoms of poliomyelitis in humans and monkeys. Bull[43] collected streptococci from the tonsils of 32 poliomyelitic cases and injected these organisms into various animals including guinea-pigs, cats, dogs, rabbits, and monkeys. The lesions produced by the streptococcic cultures were in no way identical to those observed in poliomyelitis, and in some cases they were similar to those resulting from streptococcic infection. Furthermore, the recovered animals showed no resistance to subsequent inoculation of the poliomyelitic virus, and their blood did not neutralize the filtered virus *in vitro*.

Kolmer, Brown, and Freese[44] isolated the streptococcic organism from the brain, cord, and various internal organs of fatal cases of poliomyelitis. It was found in only 1 of 20 anaërobic blood cultures. It could not be isolated in the cerebrospinal fluid. Growth occurred both aërobically and anaërobically, and under the latter condition the organism became small and globular and was more easily decolorized with alcohol in the Gram stain. Inoculation tests with the streptococci in rabbits and monkeys gave rise to arthritis and meningitis, but failed to produce any paralytic symptoms. Kolmer, Brown, and Freese also found that three different kinds of microorganisms, the diplococci, the diphtheroid bacilli, and the Gram-negative bacilli, might be grown in cases of acute poliomyelitis. The diplococci were present in the nervous tissue and various internal organs, as well as in 48 out of 106 cerebrospinal fluids. These organisms were Gram-positive, and when transplanted to solid media grew abundantly. Under anaërobic conditions, the growth was slow and the cocci became small and round. These organisms did not produce paralytic conditions either in monkeys or in rabbits, but they did set up inflammatory lesions. Complement-fixation tests by Freese and Kolmer showed that antibodies for these cocci might be found in the body fluids of persons suffering from poliomyelitis. Cohen, Heist, and Kolmer[45] showed that sera of convalescents and animals immunized with the diplococci contained an increased amount of what appeared to be a specific opsonin.

Other reasons for doubting the streptococcic origin of poliomyelitis are: The failure to find in paralytic cases any traces of metastatic infection and inflammation, ordinarily occurring in streptococcic infection; the lack of evidence that any streptococcic infection ensures permanent immunity; and most important is the fact that the animals, in which these organisms have produced paralytic lesions, have usually been found refractory to the virus of poliomyelitis obtained by ordinary methods. It is true that almost any organism injected into an animal will find its way into the nervous system and cause lesions, provided the animal is not killed too promptly by the organism, and that the amount of the microbic agent injected is sufficient.

More conclusive proof is required for the acceptance of the streptococcus as the cause of poliomyelitis. The weight of opinion at present is in favor of the globoid bodies.

THE TRANSMISSION OF THE DISEASE TO ANIMALS.

At the present time, the monkey is the only animal that can be inoculated satisfactorily. Other aninals, such as rabbits and guinea-pigs, which have shown a susceptibility to the disease, have not developed lesions characteristic of poliomyelitis. Attempts have been made to transfer the disease to other animals, including cats, dogs, rats, horses, and sheep, but so far it has not been possible to produce the infection.

The Virus in Monkeys.—Injection of the virus into monkeys produces lesions which are very typical of those of human cases of poliomyelitis. The monkey can be inoculated successfully in several different ways, but the results obtained by all methods are not regularly the same. Positive results are obtained more frequently when the virus is injected by the intracerebral, intraspinal, intrasciatic, intranasal, and the intraperitoneal routes. Conveyance of the disease by the subcutaneous, the intravenous, or the gastrointestinal route is much more difficult. When the virus has become adapted to the monkey, the transmission of the disease is much easier; lesions have been produced in some instances by simply painting the nasal mucosa with a highly adapted virus.

The Virus in the Rabbit.—The rabbit has not proved a favorable host for inoculation of the virus. Some experimenters, including Lentz and Huntemüller,[46] Marks,[47] and Rosenau and Havens,[48] claim to have produced the disease in rabbits by inoculation and to have transferred it successfully through several generations, but the resulting lesions were not characteristic of poliomyelitis. Other observers, among whom are Römer and Joseph,[40] Landsteiner and Levaditi,[25] Leiner and von Wiesner,[13] and Flexner and Lewis,[33] have not succeeded in conveying the disease to rabbits.

The Virus in the Guinea-pig.—The susceptibility of the guinea-pig is still in question. Neustaedter[49] reported having transferred the disease from one guinea-pig, which had apparently contracted the disease through contact with a monkey, to another guinea-pig, and back again into the monkey. Römer and Joseph[40] observed that guinea-pigs kept in the laboratory sometimes died from a paralytic disease which, upon investigation, seemed due to a filterable virus. The same observers made attempts to transfer the virus from monkeys to guinea-pigs, but were unsuccessful.

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CHAPTER IV.

PATHOLOGY.

GREAT advances have been made in the study of the pathology of poliomyelitis since the time when it was thought that the lesion was localized in the weakened muscles. Cornil,[1] in 1863, was the first to prove this early theory incorrect, and to point out that the localization of the primary focus was in the spinal cord. In 1870, Charcot[2] went a step further, and demonstrated that the pathological changes were localized particularly in the ganglion-cells of the anterior horns. Some years later, Goldscheider[3] recognized that the affection was not confined to the anterior horns alone, but that it involved the dorsal horns as well. He also pointed out the close connection between the lesion and the distribution of the central branches of the anterior spinal artery, and considered that the recovery of power was directly dependent upon the extent and severity of the vascular disturbance. Many investigators, principally Wickman,[4] Harbitz and Scheel,[5] and Strauss,[6] have presented more accurate pictures of the exact pathological conditions, and have settled definitely that it is possible for any part of the central nervous system to be involved. More recently, the dissemination of lesions throughout structures and organs other than the nervous system has been determined, a discovery which is wholly in keeping with our recognition of poliomyelitis as a general infectious process. Lesions have been noted particularly in the lymphatic system and in the parenchymatous organs.

The following description of the pathology of poliomyelitis is based particularly upon the reports of Peabody, Draper, and Dochez,[7] Vulpius,[8] and the New York City Health Department of 1917.[9]

In the modern conception of the pathology of poliomyelitis, two processes are recognized as important:

1. The hemorrhagic reaction, with edema.
2. The destruction of the anterior nerve cells.

The damage of the cells is responsible for the paralytic disturbances. Whether the injury is due to the direct action of the virus, or to toxic formations produced by it, or whether the damage is secondary to a hemorrhagic process in the interstitial tissue, has not yet been solved. Studies of the changes which take place in the nervous system suggest that the nerve injury is a secondary lesion, and is the result of disturbances in the nutrition of the nerve, the accumulation of waste products, and the pressure of edema and hemorrhage.

CHANGES IN THE ACUTE STAGE.

In the Central Nervous System.—The earliest change in the nervous system consists of an acute meningitis, resulting from the infiltration of small mononuclear cells, probably lymphocytes, in the vicinity of the bloodvessels of the leptomeninges. Incidentally, the presence of this early alteration in the leptomeninges, which, by means of the lymphatics that pass outward with the filaments of the olfactory nerve, are in direct communication with the upper nasal cavities, strengthens the evidence that the virus enters the human body through the upper respiratory tract. The extent of the infiltration is proportionate to the vascularity of the area, and is more pronounced in the floor of the fourth ventricle, in the cervical and lumbar regions, and particularly in the anterior fissure from which the larger vessels enter the cord. Smaller collections of cells are often found along the meningeal vessels located over the lateral and posterior aspects of the cord.

The vessels of the meninges are the source of blood supply to the cord, and as the pathological process advances, the perivascular infiltration follows along with the vessels and enters the cord. Thus, in the cord itself, there is formed a cellular exudate, due to hyperemia and the collection of small round cells in the lymph spaces surrounding the vessels (Fig. 1). This exudate may form a sheath and completely surround the vessels for long stretches. In some cases it may be so great that it presses on the lumen and obstructs circulation. Undoubtedly, this exudate has some effect, either toxic or mechanical, upon the intimal lining of the vessels, for hemorrhages varying in severity are frequently observed, and one of the most prominent features of most cases is the extensive edema (Fig. 2). The regions having a more abundant blood supply show correspondingly greater vascular changes. The anterior gray matter of the anterior horns, to which the blood supply is copious, shows marked lesions. The posterior gray matter is less often affected, although in some cases it may be densely infiltrated, especially in the region of Clarke's column. The white matter is very seldom affected. The tendency of the lesions to be asymmetrical

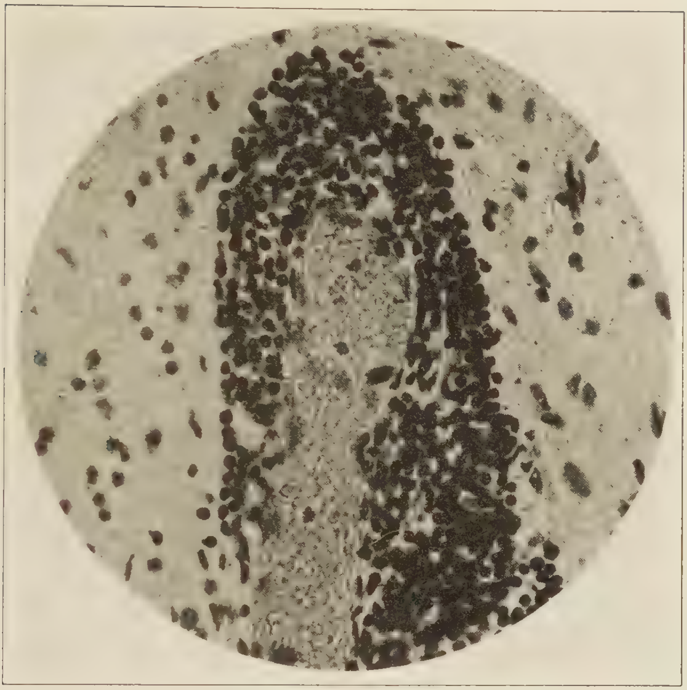

FIG. 1.—Cord. Inflammatory alterations about small vessel.

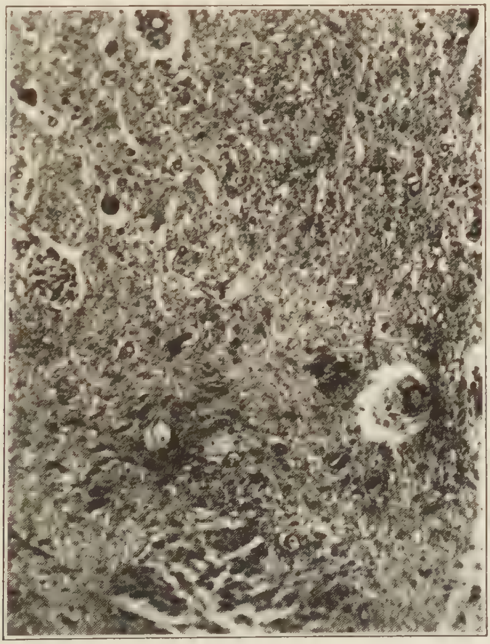

FIG. 2.—Perivascular infiltration with lymphocytosis, small hemorrhages, and apparent edema.

probably is due to the irregularity with which the vessels supplying the cord are given off at different levels.

As to the actual changes that take place in the cell, atrophy of the intracellular network of the neurofibrillæ first occurs. The body swells and becomes globular, and disintegration of Nissl's granules occurs. The nucleus may remain normal in appearance and retain its position for a considerable time. If the pathological process continues, the nucleus becomes irregular, and eventually may be entirely destroyed. The amount of destruction depends, of course, upon the severity and duration of the lesion. In the most severe types, the hyperemia, perivascular infiltration, and edema may be seen throughout the gray and white matter, but no nerve cells may be present.

In the majority of cases, there is a recession of the cell infiltration, and some of the edema and congestion disappear. As this reparative process goes on, cells that have been only temporarily under pressure will recover their function. If the damage has been prolonged or extensive, the cells may be entirely destroyed and will never recover. There are other cells that may be only slightly damaged, and with proper care these will regain their power. In general, the localization of the paralysis has been found to correspond to the pathology in the spinal cord. As yet, however, no definite relation has been worked out between the variance in severity of the paralysis and the degree of damage in the cell.

Vascular and nerve-cell changes similar to those in the spinal cord are found in the brain-stem, cerebrum, and cerebellum, but the lesions are less extensive. The vessels most infiltrated are those lying just beneath the fourth ventricle; the deeper-lying ones tend to be less involved. The vessels of the cerebrum and cerebellum may present hyperemia and some cellular infiltration, but the damage is seldom severe enough to produce motor symptoms. The cerebrum is affected much less frequently than other parts of the nervous system. The cerebral meninges, as a rule, are not involved. The medulla and pons usually show a slight degree of involvement in the form of a marked cellular exudate and many hemorrhages.

Changes similar to those in the spinal cord also take place in the posterior root ganglia. First, an infiltration of small mononuclear cells proceeds from the pial investment, and from the blood-vessels which may arise either through the general blood or through the inclusion of the vessels in the infiltrative process within the septa of the nerve roots. The ganglionic nerve cells may be destroyed by focal secretions of mononuclear cells or by necrosis. It is probable that the involvement of the sensory ganglia and the pinching of the posterior roots account for the presence of pain in the acute stage. The cellular infiltration of the cells along the nerve roots may also be responsible for some of the pain.

In a postmortem examination, the meninges appear injected and edematous. On section, the surfaces of the brain and cord seem moist, soft, and edematous. In many cases, the gray matter of the cord is so swollen that it bulges above the level of the surrounding white matter and is sharply demarcated. The gray matter often assumes a grayish-pink hue, and in severe cases is very red. Frequently, tiny hemorrhagic areas may be distinguished in both the gray and white matter. The cerebrospinal fluid usually flows under slightly increased pressure and shows changes. These changes are described in the section on Lumbar Puncture. In some cases, the cord may appear normal to the naked eye, and cerebral changes also may be invisible, but microscopic examination will reveal the presence of pathological changes.

Changes in the Lymphatic System and Parenchymatous Organs.— The virus has also been found to be productive of extensive pathological changes in the lymphatic tissues and in the parenchymatous organs. Peabody, Draper, and Dochez[7] found such involvement in 11 acute cases which they examined. Peyer's patches of the intestine show congestion, some of the mesenteric glands are swollen, and there is hyperplasia of round cells, changes resembling those observed in typhoid fever. The mucosa over Peyer's patches, however, is not involved. Lymph nodes in other parts of the body, such as the substernal, bronchial, cervical, axillary, and inguinal glands, and the tonsils and other lymph structures, are somewhat affected. The spleen is considerably increased in size, and shows a varying degree of hyperplasia of the Malpighian corpuscles. The thymus also shows changes. The tonsils are found to be larger than normal in some cases.

Histological examination of the lesions in the lymphatic system shows that while some of the lymphoid nodules are apparently normal, the majority of them show a belt of lymphocytes surrounding a pale center made up largely of endothelial cells with oval vesicular nuclei. The better preserved cells are decidedly phagocytic and show traces of necrotic cells. Usually the degenerating cells are lymphocytes, although the endothelial cells are also found broken down. Polymorphonuclear leukocytes are present in extensive necrotic areas.

In the lymph sinuses a great number of phagocytic endothelial cells have been found, many of which are of large size and contain necrotic fragments of nuclei, lymphocytes, or numerous red blood corpuscles. There is also an extensive proliferation of endothelial cells in the lymph sinuses, in contrast to the necrosis found in the centers of the lymphatic nodules.

Of the parenchymatous organs, the liver seems most markedly affected. Necrotic areas, varying in extent from the involvement of one or two cells to a considerable area, are present. While these areas are chiefly associated with the portal vein, they show some connection with central or sublobular vessels. The lesions may be round or oval in shape, or extend in long slender projections from the chief area of involvement.

The kidneys show a cloudy swelling, congestion, edema, and beginning degeneration. In severe cases, an acute nephritis may be present. Clinically, cases supposed to be suffering from retention, on catheterization, have been found to be suffering from anuria, due, perhaps, to the action of the virus on the secretory cells of the uriniferous tubes, or to total inhibition of the centers for innervation of these cells (Frauenthal[10]).

The heart and lungs show no marked changes, except in cases of respiratory involvement in which the lungs present acute edema and congestion (New York Monograph[9]).

CHANGES IN THE CHRONIC STAGE OF POLIOMYELITIS.

As soon as the cells become necrotic, neurophages enter and carry away the débris. There exists a proliferation of neurogliar tissue, which replaces the destroyed nerve structures. As time goes on this neuroglia contracts and forms scars. These are seen in the cord in old-standing cases. The exact source of these cells is not known; some observers believe that they are derived from the white cells of the blood; others that they are produced by a proliferation of the fixed cells of the adventitia of the blood cells; others that they are derived from the neuroglia of the central nervous system (New York Monograph[9]).

The atrophy found in the cord will depend upon the amount of destruction. Often the wasting is limited to one lateral half of the cord, or to one anterior horn. The ganglion-cells may be degenerated beyond recognition or may be entirely absent (Figs. 3, 4, and 5). The fibers of the anterior horn are also degenerated, and the nerve trunks atrophied.

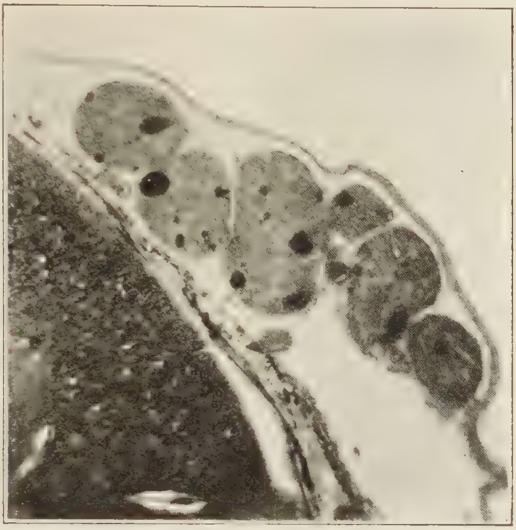

Fig. 3.

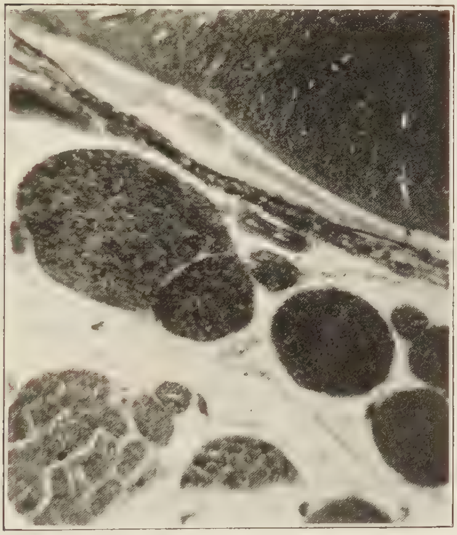

Fig. 4.

Figs. 3 and 4.—Anterior nerve roots from the lumbar region stained by Weigert's myelin sheath stain. The lack of staining is due to loss of myelin. Contrast with the posterior roots (Fig. 4) which are normal and stain black.

As a result of the destruction of the nerve cells in the anterior horn the peripheral nerve atrophies, and the muscle supplied by that nerve degenerates. The degree of degeneration depends upon the amount of nerve supply cut off. The atrophied fibers are easily recognized by their yellowish-white color. In some cases, complete degeneration is brought about by a fatty deposit around the atrophied muscle. Occasionally, muscles subnormal in size present apparently normal fibers, red in color; these fibers either have been overstretched or have atrophied from disuse.

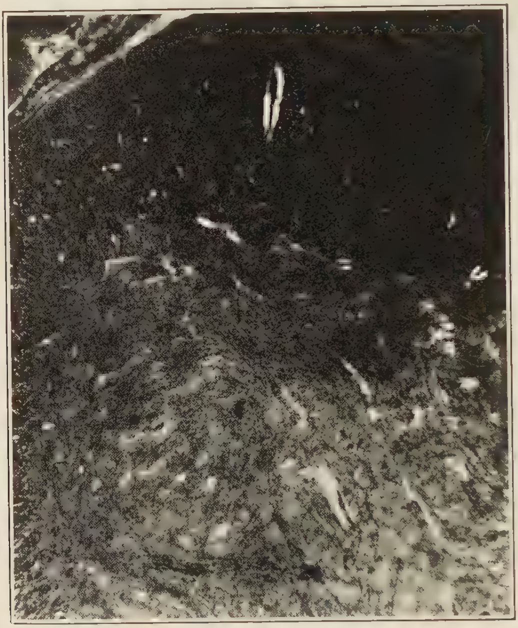

Fig. 5.—A late case of poliomyelitis, showing an anterior horn from the lumbar region of the spinal cord, stained by Weigert's myelin sheath method. Note the almost complete absence of motor cells.

Tendons also atrophy from disuse and become small, weak, and likely to yield at the point where they are intersected by the degenerated muscle.

Bones are often involved in the pathological process. The long bones of a paralytic limb are more slender than the normal bones. The outer layer is rarefied, and the medulla is reduced in quantity, as demonstrated at autopsy and in radiograms. Deformity often develops with age. Shortening may occur from lack of use and involvement of the nutritive centers. The capsule and ligaments become stretched, and the joints become hypermobile and occasionally are dislocated.

PATHOLOGICAL CONDITIONS OF THE NOSE AND THROAT.

If the nose and throat are the portals of entry of the virus, one naturally is interested in the pathological condition of these organs. A special study of 2000 cases of poliomyelitis was made by laryngologists in the epidemic of 1916 in New York City, to determine the condition of the nose and throat. They found that a large number of children had either diseased and hypertrophied tonsils and adenoids, or both, and a similar number showed hyperemia of the nasopharynx and throat. Only a small number of all the cases that had previously had their tonsils and adenoids removed were affected with poliomyelitis, and in this group, the percentage of recovery was higher than in the unoperated group.

In another series of 1404 cases, it was found that those whose tonsils had been operated did not develop the disease, in spite of the fact that in 18 instances cases developed in the same family, and in 93 instances cases occurred in the same house (New York Monograph[9]).

Further studies will be necessary before any definite conclusions can be drawn as to the presence of pathological conditions in the nose and throat in poliomyelitis.

AN AUTOPSY.

Russel[11] published an excellent report of an autopsy in the *Montreal Medical Journal* for July, 1910. The patient had died of respiratory failure. The autopsy was performed ten hours after death. The brain and cord were removed. The dura appeared normal, and no congestion appeared in the cord or brain. The brain was enlarged, and its surface had a slightly bluish-gray color. The superficial vessels were not particularly engorged. On section, the capillaries of the cortex were seen to be somewhat engorged in places. Other organs were examined *in situ* and showed no abnormality.

Cord.—On palpation the cord felt extremely hard over the lumbar and dorsal regions. The whole cord from the lower end of the cervical enlargement to the sacral segments had the appearance of having been wound carelessly and tightly with fine thread, showing everywhere little irregular ridges and bulges, which were evidently the swollen cord structure bulging through the strands of pial tissue. On section of the cord, the anterior horn region looked softened, swollen, and almost diffluent, particularly in the lumbodorsal region and to a less degree in the lower cervical section.

Second Sacral Segment.—Microscopically, there was infiltration of the meninges, with round cells all about the cord, but not affecting nerve routes. The superficial vessels were congested and surrounded by lymphocytic exudation. Those of the cord substance were similarly congested and showed the same surrounding round-cell exudation. Infiltration of the gray matter was also present. Many nerve cells had disappeared, but a few remained and retained their normal appearance. The congestion and surrounding exudation were quite general, affecting those vessels coming in from the periphery of the cord as well as the branches of the anterior spinal artery.

Fourth Lumbar Segment.—Slight meningitis and marked congestion of the vessels of the substance of the cord. with occasional small rupture and hemorrhagic extravasation, were present. There also existed perivascular exudation of lymphocytosis, and round-cell infiltration of the gray matter. The ganglion-cells had entirely disappeared.

Ninth Dorsal Segment.—The same conditions as above were found. Only a few nerve cells remained on the left side in Clarke's column.

Eighth Cervical Segment.—The infiltration, which was less, followed the course of the vessel into the left anterior horn; the vessel was congested, but showed no evidence of thrombosis. The congestion of the vessels in the substance of the cord was general, but the perivascular round-celled infiltration was by no means marked, nor was the infiltration of the gray matter extreme. None of the ganglion-cells was recognizable.

Fifth Cervical Segment.—The fifth cervical segment presented a picture almost identical to that just described, except that there were one or two ganglion-cells still recognizable in either horn.

Second Cervical Segment.—A similar condition was found here, save that the round-celled infiltration was again more marked, especially around the vessels and in the gray matter.

Lower Medulla.—Meningeal infiltration was present, and there was marked generalized perivascular lymphocytic exudation. The nerve cells, however, appeared normal.

Right Postcentral Convolution.—The right postcentral convolution showed marked congestion of the vessels of the meninges with surrounding exudation of the lymphocytes; the meninges showed a slight degree of infiltration; the cortex appeared nearly normal.

Right Olfactory Lobe.—Nothing abnormal appeared here, although the vessels of the meninges were congested and showed the usual lymphocytic exudation.

The posterior ganglia were not examined.

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CHAPTER V.

SYMPTOMATOLOGY.

INCUBATION PERIOD.

Varying opinions have been expressed as to the duration of the incubation period. In experimental observation of monkeys. Flexner[1] found that the period varied from two to thirty-three days, with an average duration of about nine days. In man, Wickman[2] estimated the incubation stage from one to four days, basing his opinion on the time intervening between the development of two cases in the same family. Müller[3] believed that the average period was about one week. Other writers have rated it from twelve hours to thirty days. Shaw,[4] in a study of 756 cases, found that the incubation stage was comparatively short, that is, from three to ten days. Until recently, the period was generally considered to be under eight days. A late investigation by Aycock and Eaton,[5] however, tends to show that the period is of longer duration, and averages fourteen days. This conclusion was reached from a study of the time interval between multiple cases in 253 families. From this investigation, it was also inferred that the disease was most infectious within the first three or four days of the acute stage.

PREPARALYTIC PERIOD.

The onset of poliomyelitis is accompanied by various symptoms. In some cases they may be so slight as to be overlooked, and the paralysis is the first manifestation recognized. Occasionally, a partial or complete loss of power in a muscle group is noted in older children who have no history of having suffered an illness in childhood. The symptoms are complex, with a tendency for one symptom or set of symptoms referable to the throat, gastro-intestinal tract, or to other parts of the body, to predominate in a given epidemic. The onset of the symptoms is usually sudden, although a gradual development of the disease is not unknown. In general, while there is nothing pathognomonic about the syndrome of poliomyelitis to distinguish the onset of this disease from that of many other infectious diseases of childhood, still the clinical picture during the early hours of poliomyelitis presents certain definite features that help to distinguish the disease. The position that the patient assumes to protect himself from pain, the mental condition, the glazed sclera and cornea, and the rigidity of the posterior muscles of the neck to prevent anterior flexion of the spine are signs that are particularly valuable in making a differential diagnosis.

Draper[6] has divided the symptoms of the preparalytic stage into the general systemic and meningitic forms. These symptoms may run a continuous course until paralysis sets in, or may abate before paralysis develops. Other cases present two periods of illness; the child appears to recover from the first symptoms of a general systemic infection, and after a remission, which usually lasts a day or two, suffers symptoms referable to the cerebrospinal tract. Draper gives the interesting term "dromedary" to this type.

Systemic Symptoms.—*Fever.*—Of all the symptoms fever is the most constant. Afebrile attacks have been reported, but it is possible that the fever was so slight and of such short duration that it was overlooked. The temperature usually varies from 100° to 105° F., but cases of 107° F. have been observed. The elevation of temperature is no criterion for judging the subsequent course of the disease, as cases with a high temperature may recover entirely, and those with a slight temperature may develop severe paralysis.

The high temperature is usually of short duration, lasting from one to six days. It may, however, continue abnormal for ten days, lasting into the paralytic stage. It may subside very suddenly, or abate gradually. In some cases it may drop to just above normal or slightly below normal, where it may remain for a few days or even two to three weeks. In the dromedary type the temperature may return to normal, and then after four or five days rise again.

The pulse varies with the temperature in the febrile period, there being a rise of about 10 points to every degree of fever. The pulse may continue high for some days after the temperature has dropped. The respiration rises and falls with the temperature, except when the chest muscles are involved.

Pain.—Pain in some part of the body is one of the most marked features of poliomyelitis. It may be spontaneous; it may be experienced only upon manipulation or pressure; or it may exist in the form of a general hyperesthesia of the skin. Spontaneous pain varies in degree; some patients suffer little pain, while others suffer intensely. Pain on motion or on being handled is often so intense that the child lies very quietly and resents any disturbance. Pain is elicited in some cases by gently squeezing the muscles.

In some cases pain lasts only a few seconds; in others it persists for days or even weeks. When the paralysis clears up quickly, the pain disappears with the return of power. but when paralysis persists, the pain may last for several weeks.

The pain may be localized in the extremities, in the back, along the nerve tracts, or it may extend over the entire body. Occipital headache, due to hyperemia of the cerebral vessels, is often experienced, and next to fever is the most common symptom of the preparalytic stage. It is usually of moderate intensity, but at times it may be severe. Pain in the back of the head and neck, particularly on bending the head forward, is very frequent and is a suggestive symptom. The pain in the neck is usually accompanied by rigidity of the posterior muscles, which is easily recognized on an attempt to flex the head anteriorly. This rigidity is one of the last of the acute symptoms to disappear.

Hyperesthesia, due to the early involvement of the posterior root ganglia, is a prominent symptom of the preparalytic stage. The slightest touch or even the pressure of the bedding will often elicit pain. The tenderness is usually present along the spine and down the legs, but it may extend throughout the body. It is the opinion of some writers that the hyperesthesia is more marked in the parts that later become paralyzed. Tenderness may persist weeks or even months and often leads to an incorrect diagnosis.

Gastro-intestinal. — Gastro-intestinal symptoms are common. Nausea and anorexia are very frequent. Vomiting also occurs, but as a rule it is not so persistent or so severe as in meningitis; in exceptional cases it may be so marked as to suggest acidosis. Diarrhea has been a constant symptom in some epidemics; Krause[7] found that it occurred in more than two-thirds of the cases in the Westphalia epidemic. Lack of exercise may cause constipation, but it is usually very slight.

Bladder Disturbance.—Retention of the urine occurs in a small proportion of cases. Such disturbance yields readily to palliative measures.

Sweating.—Profuse sweating, particularly in the severer cases, is not uncommon. Müller[3] considered sweating to be one of the prominent symptoms of the early stage, since he observed it in 75 per cent of his cases. Sweating may be limited to local areas, as to the face, or even to one-half the face. It may be moderate, or as marked as the colliquative sweats experienced in typhoid fever. The sweating ordinarily disappears upon recovery, but it may persist for a long time. Since sweating is likely to accompany

any fever, it should not be considered a symptom peculiar to poliomyelitis.

Eruptions.—Eruptions do not seem to be a frequent symptom. Some observers have reported cases of erythematous rashes on the neck and chest and occasionally on the trunk. These rashes may be due either to sweating or to a toxemia that develops from the infected intestinal tract. Such rashes, however, are found in cases of fever of any origin, and are not peculiar to poliomyelitis. Herpes labialis has been noted in rare cases. Desquamation has been observed, the scales being most pronounced on the neck and chest.

Respiratory Tract.—The involvement of the respiratory tract has been found to vary greatly; in some epidemics respiratory symptoms have predominated, in others, few have been noted. Regan,[8] from a study of 800 cases in the 1916 New York epidemic, found that congestion of the throat was an almost constant feature of the acute stage. Usually congestion was limited to the faucial mucosa and the pharynx. The uvula was often found small for the age of the patient. The buccal mucosa varied only slightly in color. The soft palate assumed a deep red color.

In a study of the nose and throat conditions of a series of 2000 cases, made by the Health Department[9] of New York City in 1916, it was found that a large number of children showed local pathology. In many cases there was an enlargement of the tonsils and adenoids, or of both. The abnormality of these structures is not necessarily indicative of poliomyelitis, since they may not have been normal before the attack. In some cases there was considerable coryza, and the tongue was often covered with a grayish coating.

In cases in which the intercostals or diaphragm are involved, bronchopneumonia or lobar pneumonia may occur as a complication.

Eye Symptoms.—The sclera and cornea of the eye are glazed, and there is circumorbital puffiness. These features are suggestive of poliomyelitis, for in other acute infectious diseases the eyes are usually bright and shining.

Vasomotor Disturbances. An alternate blushing and paling of various regions of the skin may occur in some cases. Small spots or large areas may be affected. The flushing is often only momentary.

Meningitic Symptoms.—Among the meningitic symptoms are twitchings, tremor, and convulsions.

Twitchings.—Muscular twitchings are commonly present. Colliver,[10] in particular, has called attention to a form of tremulous or convulsive movement of groups of muscles. It may affect a part or an entire limb, or it may involve the whole body.

Tremor. –A distinct tremor of the fingers, which may have diagnostic value, has been demonstrated by Heiman.[11] This tremor is noted when the fingers are outstretched, and is evidently due to the irritation of the anterior horns prior to the beginning of degeneration.

Convulsions.—Convulsions are not common, but they have occurred, particularly in the rachitic and undernourished child. They have been observed both at the onset and later in the course of the disease.

Reflexes.—Alterations in the reflexes depend upon whether the upper or lower neuron is involved. In the ordinary spinal case, the reflexes are exaggerated during the febrile stage. The hyperactivity of the patellar reflex is usually one of the first changes in the preparalytic stage, and it may be so marked that the slightest tap will elicit the knee-jerk. In the bulbar type the reflexes may remain normal or become hyperactive.

Mental Condition of the Patient.—*Drowsiness.*—Drowsiness is a marked symptom in most cases. It may last a day or two and disappear, or it may increase. When aroused, the patient is irritable and restless, plainly showing the desire to be unmolested. As soon as he is left undisturbed, he again relapses into his comatose state. The mind is usually clear in spite of the drowsiness. Occasionally the patient appears to be in a deep sleep. After three to six days the child may wake suddenly or gradually from this sleep.

Although, as a rule, the patients are stuporous, some cases are wide awake, excited, and tend to be delirious. The child is restless and irritable all the time, and not simply when disturbed. Such a condition is a cause for apprehension, as these cases usually end fatally.

PARALYTIC PERIOD.

Onset.—Paralysis usually appears during the first four days of the disease, and in the majority of cases it comes on during the second day. After the fourth day fewer patients develop paralysis, and after the eighth day there seems to be little danger of its occurrence.

As a rule, paralysis marks an end of the febrile symptoms. In cases of high cervical involvement, vomiting may continue for months. Pain in the involved area and backache persist in some cases, and are particularly noticeable on movement.

In the spinal type, the hyperactivity of the reflexes diminishes upon the onset of the paralysis, and during the paralytic stage the reflexes disappear entirely. In the bulbar type or the meningitic form, the hyperactivity of the reflexes may continue into the paralytic stage.

There are some cases in which the paralysis comes on without any apparent preceding symptoms. A child may be put to bed apparently in good health, and the next morning a limb is found to be paralyzed. In infants, paralysis may not be noticed until the child is bathed. It is possible, of course, that the premonitory symptoms may have been overlooked by the mother. In older children great fatigue may immediately precede the onset of paralysis; a child, while playing in the yard, may become very tired, and a short time later is unable to move his legs. Other children may stumble and fall when paralysis begins to take place. In rare cases there is a progressive muscular weakness, which at first involves only part of an extremity, then gradually affects the whole limb, and finally extends over the entire body. This progressive type is usually fatal.

Recognition of the Paralysis.—As a rule, the clinical picture is very suggestive at this period. The position of the child is one of self-protection from pain. Usually he lies on one side, with the head thrown slightly backward and the legs flexed. If there is no meningeal involvement, the child lies on his back with the legs crouched in a frog-like position. In spite of his comatose state, the patient seems to be aware of any attempt to disturb him.

In older children, the localization of the paralysis can be revealed immediately by examination, unless, of course, the child is suffering extreme pain and cannot be persuaded to move his limbs. In younger children it is often more difficult to locate the paralysis, and manipulation, pinching, or pricking the muscles with a sharp instrument may be necessary to determine its extent. In very young children, the site of the paralysis may be established by lifting the limbs and letting them drop.

During the early paralytic stage the paralysis is usually widespread, owing to the pressure on the central nerve cells from edema. There may be complete loss of power in the legs, arms, back, neck, and intercostal muscles. Aside from the spinal paralysis, the type with which we are most familiar, there are some cases in which the lesions seem localized in a particular section of the cord, and give rise to a special variety of symptoms. Such conditions have led to the classification of the paralysis according to the various symptoms. These different groups will be found discussed in the Chapter on the Classification of the Types of Paralysis.

Stage of Recovery.—As the edema in the brain and cord becomes absorbed, the pressure on the motor cells is removed, and the muscles dependent upon these cells for their innervation resume function before any material changes have taken place. As a rule, the distal muscles recover before the proximal. The upper extremities are more likely to recover from paralysis than other parts of the body. Paralysis may persist in the lower leg and in the abdomen.

In practically all cases, recovery begins within the first two weeks, although in exceptional cases improvement may not be noted for several weeks. The improvement may be rapid at first and slow later; it may be gradual; or it may be rapid for several weeks and then stop. Mild cases may clear up entirely within a week. When improvement is rapid within the first few weeks, the outlook for ultimate recovery is favorable. Complete recovery is rare in cases in which paralysis has existed for a month or more. Power may continue to return for six months, and at the end of this period the true extent of the residual paralysis can be judged. Some observers believe that power continues to return during the first two years, but in the opinion of the writer, the improvement in a muscle after six months is not due to the spontaneous recovery of a paralyzed area, but rather to the restoration of power in a weakened muscle brought about by correction of a deformity that has prevented the muscle from recovering previously.

During the early part of recovery, vascular changes occur, their severity depending upon the extent of the paralysis. One of the most constant symptoms is coldness of the extremities. If not properly protected, the limb may become cyanotic and mottled. At one time it was not unusual to see obstinate ulcers as the result of the breaking down of the skin, but owing to proper treatment, such conditions are now rarely observed.

Muscle atrophy begins within the first two weeks. The deltoid atrophies more quickly than any muscle, and the prognosis as to recovery in the shoulder is particularly unfavorable if paralysis persists.

Distribution of Paralysis.—The final localization of the paralysis varies exceedingly. A particularly striking feature is its unsystemic distribution; the most peculiar combinations may occur, such as a paralysis of a peroneal muscle and a deltoid, or involvement of the muscles of one leg and the face. The loss of function is more frequently partial than total, but cases of complete paralysis are more common than is ordinarily supposed.

The relative involvement of the different extremities and muscles has been a subject of much study. Lovett,[12] in particular, gave attention to this phase of the work and studied several series of cases to determine the local distribution of paralysis. The following table, based upon 718 cases, was prepared by Lovett and Lucas.[13]

Both legs	130
Right leg	216
Left leg	239
Right arm	5
Left arm	5
Both arms alone	0
All four extremities	3
Arm and leg, same side	15
Arm and leg, opposite side	7
One arm, both legs	2
Abdomen, with other paralysis	6

Paralysis of the Lower Extremities.—The lower extremities are affected more often than other parts of the body, owing to the fact that their nerve plexuses originate in the lower lumbar region, where, because of its extensive vascularity, the pathological lesions are frequently located. Paralysis of one leg is the most common involvement. Next in frequency is loss of power of both legs. Paralysis of the lower extremities is often accompanied by a loss of power in other parts of the body. When one leg is involved, it is common to find the muscles of the other leg somewhat weakened.

As to the frequency of involvement of the different muscles in the upper leg, the quadriceps femoris is most often paralyzed, and in the lower leg, the gastrocnemius, the tibialis anticus, and the peronei are frequently involved. The flexors of the foot and the extensors of the toes are next frequently affected. If the flexors of the toes are paralyzed, they recover before any other muscles. The tibialis anticus and the gastrocnemius are the muscles of the leg found paralyzed most commonly without loss of function occurring elsewhere (Lovett[12]).

The condition of the reflexes is interesting. Regan[14] found that the patellar reflex was changed in various ways in 81 per cent of a series of 818 cases. It was found absent in most cases. When the paralysis was bulbar in type, the reflex was exaggerated. In rare cases there was an absence of the knee-jerk on the paralyzed side and an exaggerated response on the healthy side. Regan found that the plantar reflex was changed in 41 per cent of 643 cases. In meningitic types an exaggerated response may persist into the paralytic stage. Often the gradual reappearance of the plantar reflex is the first sign of improvement in cases of marked paralytic involvement.

Paralysis of the Upper Extremities.—Next in frequency to the paralysis of the lower extremities is the involvement of the arms, which results from impairment of the innervation of the fifth cervical to the first thoracic segment. In most cases the paralysis is unilateral and is accompanied by loss of function in the leg. Paralysis of both arms is usually associated with involvement of other muscles. Loss of function is most frequent in the shoulder muscles, particularly in the deltoid. The frequency of involvement diminishes from the shoulder to the distal parts. The flexors of the fingers are seldom affected; if paralyzed, they show a tendency to regain power quickly. Paralysis of the abductor of the thumb is very common.

Paralysis of the Back and Neck Muscles.—Paralysis of the back muscles or of the neck muscles may occur alone, or both groups may be involved in the paralytic process. These muscles are more frequently affected in severe paralysis. The presence of paralysis of the back muscles is easily overlooked during the early stage when the child is so ill that it is difficult to make examination. Undoubtedly, the back muscles are involved more frequently in the early stage than is generally recognized.

If the paralytic involvement of the back muscles is bilateral, the child has no control over his body and cannot assume an upright position. If paralysis is unilateral, the child may be able to sit up, but unless precaution is taken to prevent deformity, a marked scoliosis may result. Many cases of paralysis of the back muscles recover. Residual paralysis, however, is not uncommon and undoubtedly is a factor in the development of scoliosis.

Paralysis of the neck muscles is recognized from the asymmetrical position of the head. If the anterior muscles are affected, the head falls backward; if the lateral muscles are involved, the head falls to one side or the other; and if all the neck muscles are paralyzed, the head remains in the position in which it falls. Paralysis of the anterior muscles is very common, and Peabody, Draper, and Dochez[15] have described the following important diagnostic sign in connection with it. If the child is raised up by placing the hands under its shoulders, the head falls back. If told to raise its head, the child may be able to do so for a moment, but the head will then fall back and cannot be raised again.

It should be borne in mind that the child may hold his head backward because this position is more comfortable, and there may not necessarily be any loss of function. Neck paralysis usually clears up, and it is only in the severer type of case that a permanent weakness remains.

Abdominal Paralysis.—Loss of power in the abdominal region is not uncommon. The involvement is usually found in combination with paralysis of the back or the extremities, particularly the legs.*

Paralysis of the Intercostals.—The muscles of respiration are affected only rarely, owing undoubtedly to the scanty blood supply of the thoracic segment of the spinal cord. Involvement of these muscles most often occurs in severe cases of the ascending or descending type, which terminate fatally. Some cases of respiratory involvement do recover either completely or with a permanent partial or complete paralysis of the intercostals (Figs. 6 and 7).

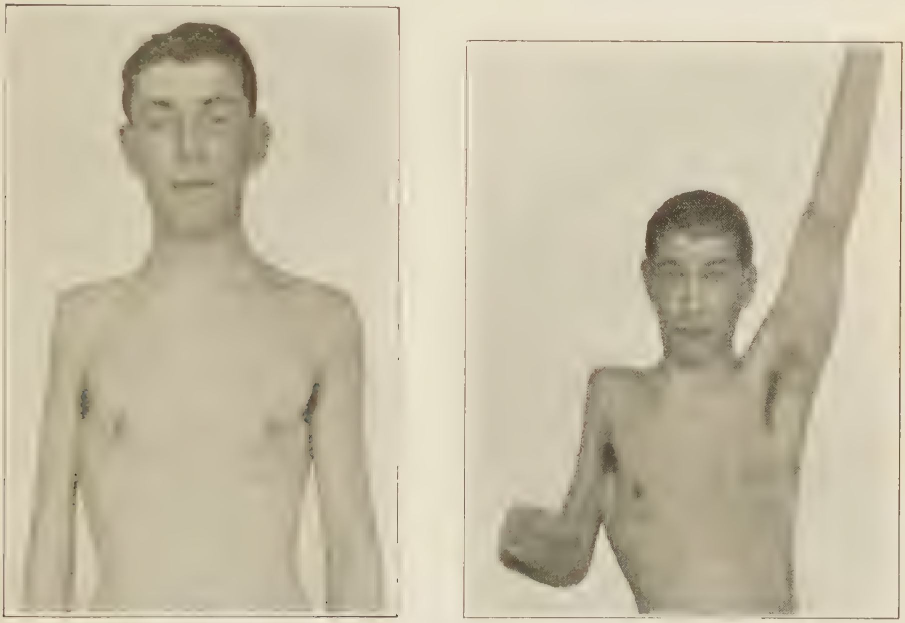

FIG. 6. FIG. 7.

FIGS. 6 and 7.—Paralysis of the intercostals and the deltoid of the right arm.

In intercostal involvement, the respiration is entirely diaphragmatic in character; on inspiration the thorax moves very little, and its motion is downward and backward, instead of forward and upward as normally.

It may be difficult to determine partial paralysis of the respiratory muscles, as some children normally have abdominal respiration.

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* For complete description, see Trunk Paralysis.

The cases of extensive involvement, however, are easily recognized.

Draper[6] has called attention to the very striking condition of the sensorium of a child suffering from respiratory involvement. While infants tend to be in the usual stuporous state, older children seem to sense the approaching danger. They are usually wide-awake, alert, with every effort concentrated on the process of breathing. As respiration grows weaker, the mind becomes dull, and the child gradually sinks into unconsciousness.

Paralysis of the Diaphragm.—Paralysis of the diaphragm rarely occurs, as the phrenic nerve, which innervates the diaphragm, rises from the upper part of the cervical spine, a section rarely affected in poliomyelitis. The diaphragm is liable to be involved in fatal cases, usually being the last muscle to be attacked before death occurs. Cases have been seen, however, in which paralysis of the diaphragm cleared up entirely, even after several weeks.

Paralysis of the diaphragm is accompanied by dyspnea, and is easily recognized. The abdomen retracts with each inspiration instead of pushing forward as under normal conditions. The patient is usually alert and fearful of any attempt to disturb him.

Paralysis of the Bladder.—The bladder is only rarely involved. Le Boutillier[16] found disturbances of the bladder in only 2 per cent of a series of 768 cases. This trouble usually occurred between the second and fifth days of paralysis and disappeared within forty-eight hours. As a rule the condition cleared up rapidly without catheterization, and no cystitis followed. Undoubtedly the bladder disturbances are similar to those in other acute febrile conditions, and are not the direct result of affection of the spinal cord.

Sphincter Paralysis.—Disturbances of the bladder and rectal sphincters are rare. In severe cases of paralysis, the child may have little control of the bowel, but when the sphincter recovers, power is soon regained.

Facial Paralysis.—Of all the cranial nerves, the facial is most frequently affected. The paralysis may be so slight that it is not detected at all, or it is recognized only when the face is in a certain position.

Fraser[17] called attention to the frequent involvement of the facial muscles in the common type of paralysis. Of 90 cases, 31 had weakness of the muscles of the face; in 5 of these cases, it was the only paretic symptom, and in the remaining 26, other parts of the body were affected. In 1 case, the weakness was bilateral, in 7 cases, unilateral, and in the others there was a definite asymmetry involving a weakness of the whole of one side or of the upper or lower parts of the facial musculature. The symptoms of facial paralysis vary; some patients cannot wrinkle the forehead, others cannot shut the eye, others complain of the mouth drooping on one side. Patients have difficulty in pronouncing the labial consonants, and in keeping food from collecting in the paralyzed cheek during mastication.

When the trigeminal nerve is entirely paralyzed, there is loss of sensation on the corresponding side of the face and scalp, as far as the angle of the jaw. There is anesthesia of the cornea and conjunctiva, and of the mucous membrane of the corresponding side of the nose, mouth, and soft palate, as far back as the circumvallate papillæ. There is impairment of the taste in the anterior two-thirds of the tongue.

Some cases of facial weakness recover in a few weeks, others improve more slowly, and still others are left with a permanent paralysis.

Ocular Paralysis.—The eye muscles generally escape paralysis, but ocular manifestations have been noted. Complete oculomotor paralysis was observed by Wickman,[2] and 1 case was seen by Ruhräh and Mayer[18] in the New York epidemic of 1916. In ocular paralysis the patient has no control over the movement of the eyes and must depend upon the movement of the head to see an object.

The paralysis of the external rectus muscle, causing a convergent squint, is most common. Medin[19] noted 6 cases of paralysis of the abducens. Cases were also observed by Wickman.[2]

As regards the involvement of the optic nerve, Peabody, Draper, and Dochez[15] found no abnormalities in the examination of many cases. In some instances photophobia may be present during the first days of paralysis. Blindness rarely occurs, and when present, disappears quickly. The patient may have double vision, and in his attempt to obviate the diplopia, ocular congestion results.

Gowens[20] has collected the material that has been published upon the observations of ocular manifestations. In paralysis of the oculomotor nerve, ptosis may be present. The occipito-frontalis is overactive and the eybrow on the side involved is higher than that on the normal side. Divergent strabismus may result, and the patient cannot move the eye upward, directly downward, or directly inward. Of 752 cases in the New York epidemic of 1907, 18 had paralysis of the eyelids, and 26 had strabismus. The pupil is dilated, owing to paralysis of the sphincter, and it does not react either to light or accommodation.

If the superior obliquus is paralyzed, the movements of the eye are but slightly affected. The patient, however, suffers a diplopia when he looks downward and outward. In going down stairs, he experiences dizziness and holds the head forward and toward the well side.

In paralysis of the trigeminal nerve, which contains the sensory fibers of the eye, there is anesthesia of the cornea and conjunctiva. In the prodromal period of the Hesse-Nassau epidemic of 1901, 50 per cent of the cases showed a conjunctivitis as the first symptom.

Paralysis of the Mouth and Throat.—When the glosso-pharyngeal nerve is involved, there is difficulty in deglutition and a loss of taste and of common sensation on the posterior third of the tongue on the affected side and on the soft palate. Sensation is also lost in the upper part of the pharynx.

When the hypoglossal nerve is involved, the lesion is usually unilateral. There is atony of the longitudinal muscles of the paralyzed side, and when the tongue lies quiet in the mouth its apex deviates slightly to the non-paralyzed side. As a result of atony of the hypoglossal muscle, the base of the tongue usually rises higher on the paralyzed side than on the sound side.

Movements of the tongue are diminished; it is difficult to remove food lying between the teeth and the cheek. On thrusting the tongue out, it deviates to the sound side. Speech disturbances are present, especially in pronouncing the labials and linguals (Jelliffe and White;[21] Ruhräh and Mayer[18]).

Nose and Ear.—No involvement of the olfactory nerve seems to have been reported, but it is posible that no direct tests have yet been made to determine its condition. In the few cases in which the ear was examined, it was found to be normal, or it showed a slight insertion of the handle of the hammer (Ruhräh and Mayer[18]).

Complications.—Toxemia frequently occurs in severe poliomyelitis. In intercostal paralysis, bronchitis, and occasionally lobar pneumonia may occur as complications.

Development of Deformity.—In partial paralysis, deformity will develop from the effects of gravity, unopposed muscle pull, and stretching of the paralyzed muscles, unless measures of prevention are taken. The presence of deformities interferes with the return of muscle power and function. The subject of deformity is very important; it will be found discussed in detail in the chapter on General Treatment of Poliomyelitis.

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CHAPTER VI.

CLASSIFICATION OF THE TYPES OF POLIOMYELITIS.

Early in the study of poliomyelitis it was observed that the infection frequently presented symptoms of one definite type, due undoubtedly to the localization of the infectious organism in a specific part of the central nervous system. This observation led to the inference that a classification, based upon the various types of symptoms or upon the pathological anatomy, or upon a combination of the anatomical lesions and clinical manifestations, would be a means of grouping our knowledge of the disease and simplifying our study of it. No classification, however, was found suitable for application to all cases. Indeed, it would be very difficult to make a rigid differentiation between the various types of the disease, because the clinical symptoms do not always correspond closely to the pathological findings, and the lesions are often so widely disseminated that it is necessary to classify a case partly under one group and partly under another.

Wickman[1] suggested the first classification of any importance. He recommended the following divisions:

1. The spinal poliomyelitic type.
2. The form resembling Landry's paralysis.
3. The bulbar or pontine type.
4. The encephalitic form.
5. The ataxic form.
6. The polyneuritic type (resembling neuritis).
7. The meningitic type.
8. The abortive type.

While this classification includes nearly all the different forms of the disease, its value has been questioned because of its complexity and its construction on both pathological anatomy and clinical grounds. Groupings have been suggested to simplify that of Wickman. Müller,[2] using as a basis the anatomical pathology, divided the cases into:

1. The spinal form.
2. The bulbar form.
3. The cerebral type.

4. The abortive type.

Koplik[3] described the following types:

1. The abortive.
2. The bulbospinal.
3. The cerebral and meningeal.
4. The bulbopontine.

This classification is based upon the conception of the disease as an acute infection involving certain parts of the general nervous system and stopping there, or attacking the entire cerebrospinal axis in one stroke.

Draper[4] has used as the criterion for classification the two distinct phases of the disease, one, of a general systemic nature, and the other a central nervous disorder. He has separated the cases into three distinct groups:

1. The "dromedary," in which there is an interval of well-being between the general systemic phase and the onset of the nervous symptoms.
2. The straggling group, in which the course of the two phases of the disease is continuous.
3. The sudden onset series, in which the nervous disorder is manifest at the beginning of the disease.

The New York Health Department of 1917[5] suggested the following classification:

1. Non-paralytic or abortive type.
2. Ataxic type, including cases with nystagmus in which there are anatomical changes in Clarke's column, the cerebellum, or the intervertebral ganglia.
3. Cortical type, in which the lesion is in the upper motor neuron, and a spastic paralysis results.
4. Ordinary spinal or subcortical type, in which the lesion is in the lower motor neuron, and a flaccid paralysis results.

Ruhräh and Mayer[6] subdivided the last group into the meningitic, bulbar, bulbospinal, spinal, polyneuritic, and ascending forms.

The division as suggested by Peabody, Draper, and Dochez[7] is simple and attempts to reconcile the outstanding clinical phases with the predominant pathological lesions. They recognize three groups: The non-paralytic or the abortive, and two groups of paralytic cases. Under the first division of the paralytic group are listed those cases in which the primary lesion is located in the lower motor neuron, resulting in the flaccid paralytic conditions most ordinarily encountered, such as paralysis of the muscles supplied by the cranial nerves, or paralysis of the muscles of the extremities and trunk. Included in the second group are the cases in which the upper motor neuron is involved, giving rise to an encephalitic or cerebral condition, characterized by a spastic paralysis. But even this division does not form the basis for rigid differentiation, as cases have been described in which both a spastic and flaccid paralysis have occurred, due to involvement of both the upper and lower motor neurons. Because of its simplicity, however, the classification finds more favor than the other groupings, and the writer has used it as the basis for his description of the various types of the disease.

THE ABORTIVE TYPE.

The abortive type is one that goes through the acute stage of poliomyelitis, without sufficient involvement of the nervous system to cause a frank paralysis. Some of the cases present a muscular weakness that is suggestive of paralysis, but no true involvement is evident upon examination. It is possible that a muscle weakness or even a slight paralysis is overlooked in many cases, particularly in young children, whom it is difficult to examine.

The first report of an abortive case was included in an article published in 1894 by C. S. Caverly.[8] In a small epidemic in Vermont State, he recognized 6 cases which presented a distinct rigidity of the spinal muscles, but which did not come to paralysis. Later, Wickman[1] noted several abortive cases in the Swedish epidemic of 1905, and called attention to the fact that many cases in times of epidemic are not associated with paralysis.

It is impossible to estimate the number of abortive cases in an epidemic. Several observers place the incidence of this type at about 50 per cent of the total number of cases; others estimate it as high as 70 to 80 per cent. While it is possible that many abortive cases escape recognition, it is also true that many such cases are incorrectly diagnosed as poliomyelitis, since the occurrence of a paralyzed case influences the diagnosis of any indefinite illness that occurs within a few days of a frank case of infantile paralysis.

Variance in the symptoms of the abortive cases, corresponding closely to that found in the clinical manifestations of the acute stage of cases that later come to paralysis, was first noted by Müller.[2] Meningeal symptoms may predominate in one locality during an epidemic, while in another, definite sore throat may usher in the disease. All the various symptoms have been taken up in detail under the heading of symptomatology, but a brief review of them in this section is not amiss.

Wickman,[1] using the variance in symptoms as a basis, suggested the following classification of the abortive cases:

1. Those with the course of a general infection.
2. Those with meningeal symptoms.
3. Those with pains suggesting an influenza.
4. Those accompanied by gastro-intestinal disturbance.

To this division, Ruhräh and Mayer[6] have added those cases that begin with a definite sore throat.

The cases that present symptoms of a general infection, such as fever, vomiting, diarrhea, are most difficult to recognize. The diagnosis may be made readily only when other recognized cases of poliomyelitis occur in the same family or neighborhood. Lumbar puncture will solve the question of diagnosis in doubtful or early cases.

The cases with symptoms referable to the nervous system are more easily recognized, as they suggest at once a meningitis or poliomyelitis. Careful examination to determine the presence of any antero-posterior stiffness of the neck, Kernig's sign, Macewen's sign, Brudzinski's neck and leg sign, and finally the child's inability to prevent the head from falling backward when he is raised up, will point toward the diagnosis. Lumbar puncture is always advisable in such cases.

Any difficulty in the diagnosis of cases which have pain similar to that found in influenza may be cleared up by lumbar puncture. Such cases are rare, as the pain in influenza usually is not so severe, or of the same character, as that in poliomyelitis.

Cases with gastro-intestinal disturbances or with sore throat and coryza are difficult to diagnose before other symptoms and signs peculiar to poliomyelitis are manifest. Vomiting, with fever, does not at once suggest the presence of poliomyelitis, and if the child has suffered from such attacks previously, the physician may easily be misled in his diagnosis. A diacetic reaction in the urine will lead to the suspicion of acidosis, but any febrile condition may present a diacetic reaction to some degree.

The abortive type may clear up within a few days, the usual duration of the symptoms being from twenty-four hours to eight days. In rare cases, convalescence is prolonged, and indefinite symptoms persist for several days, or even weeks, after the onset. Transient pain, experienced particularly during the night, is one of the most lasting symptoms, but it disappears spontaneously, or after light massage of the involved part. Cramps may occur in the aching muscle. The child tires easily. One limb or muscle group may be weak, indicating that the spinal cord cells governing this part have been involved to a slight extent. Examination of the muscle power by special tests will clearly reveal any weakness and confirm the diagnosis.

TYPES IN WHICH THE LESION IS LOCALIZED IN THE LOWER MOTOR NEURON.

The Spinal Type.—*The Ordinary Spinal Type.*—This type is met with most commonly. As its various phases have been treated in detail throughout this work, only a short summary of the principal features of the spinal type will be given in this section.

The onset of the disease is usually sudden, although an insidious development is not unknown. The early symptoms consist of fever, drowsiness, sweating, headache, gastro-enteric disturbance, pain, hyperesthesia, and occasionally sore throat and difficulty with micturition or defecation. There may also be meningitic symptoms, such as muscular tremors and twitchings, with diminution or loss of reflexes. Rigidity of the neck is a most important sign indicative of the disease, and is one of the last symptoms of the acute stage to disappear. The symptoms vary in severity and duration.

In the presence of such manifestations, a lumbar puncture should be done to examine the spinal fluid, which is characteristic at this stage of the disease. The fluid ordinarily shows a definite increase in the number of cells, with a predominance of the mononuclear lymphocytes. There is an increase in albumin and globulin, and Fehling's solution is promptly reduced.

Paralysis is usually demonstrable from the second to the fourth day of the attack, after which time it is less likely to develop. The paralysis is flaccid in character. It is usually preceded by the symptoms mentioned above, but cases have been noted in which the onset of paralysis has been abrupt. A period of spontaneous recovery lasting six months to a year follows the development of paralysis. The final paralysis is always less than the initial, and is more often partial than total. A study of the involvement of the various muscles has shown that the lower and upper extremities are more often affected than other parts of the body.

If precautionary measures are not taken during this period of recovery, deformities develop as the result of disturbance in muscle equilibrium. If not corrected, the surviving muscles atrophy, and bone growth is disturbed, giving rise to more disability and to ultimate shortening.

The Polyneuritic Type.—This type, resembling a multiple neuritis, has been noted by several observers. The neuritis usually appears in the early paralytic stage when the tenderness is beginning to subside, and persists for one or two weeks. Cases have been reported in which the pain, sometimes in combination with ataxia, persisted along the nerve trunks for a longer period, but such cases are rare. In some instances the pain and tenderness may be so prominent as to cause the paralysis to be overlooked, and the case to be considered rheumatism or scurvy; in other cases the pain may cause the child to hold the limbs rigidly, and to resist any attempt to move them. This condition of spastic and flaccid weakness occurs in cases of partial paralysis, and may lead to errors in diagnosis.

The pathology of these cases has been little studied, and the exact pathological condition of the nerves is obscure. Wickman[1] found no inflammation of the nerves apparent at autopsy. It is probable that the pain is due to changes in the nerve roots, spinal ganglia, or cord.

The Meningitic Type.—Practically all cases of poliomyelitis show signs of meningeal involvement. Some cases, however, may present such a typical picture of meningitis that they can be differentiated from true cases of meningitis only by means of examination of the cerebrospinal fluid. The cases in which the meningeal irritation is prolonged resemble tubercular meningitis. Other cases may simulate cerebrospinal fever.

The meningeal symptoms may appear at the onset of the disease, or they may not set in until a few days after the general systemic symptoms. Headache, vomiting, rigidity of the neck and back, opisthotonos, delirium, coma, Kernig's sign, strabismus, tonic and clonic spasms, and fibrillary twitchings of the muscles are among the most common symptoms. There may also be vasomotor disturbances in the form of flushing and paling of the skin, and the *tache cérébrale* may be present.

Diagnosis from this syndrome is impossible before the appearance of paralysis. Even after its onset, one may not be certain of the nature of the disease, as paralysis may occur in the tubercular type of meningitis. A lumbar puncture usually removes any doubt in such cases. The differential diagnosis of poliomyelitis and the different forms of meningitis is outlined under the Section on Diagnosis.

As a general rule, the meningeal symptoms disappear within a few days, and the prognosis as to recovery is good in such cases. If the symptoms fail to clear up within a few days, the patient usually dies.

The Progressive Form.—Included in this group is the great majority of fatal cases. Death occurs from the involvement of the intercostals or the diaphragm. The affection of the respiratory centers does not necessarily indicate a fatal outcome, as sometimes cases recover with a paralysis of either respiratory group.

The paralysis may be ascending or descending in character. The former type resembles Landry's paralysis, in which first the legs, then the intercostals, arms, neck, and finally the diaphragm are involved. In most cases, the initial paralysis is more extensive, affecting both the legs and arms.

The progression of the paralysis is not uniform. In rare cases, it advances rapidly without intermission, and death occurs within a few hours or days. Generally, there is a cessation for a few hours or days after the onset, and then the paralysis again starts its upward course. Before the respiratory centers are reached, the paralysis may have ceased its progression several times. The entire course of involvement may cover six or seven days.

The first sign indicating the progression of the paralysis is laryngeal disturbance, causing hoarseness, loss of voice, and difficulty in swallowing. Then, as the respiratory centers become involved, the breathing becomes more rapid, irregular, and labored. As the diaphragm weakens, the neck muscles become more prominent, the sterno-cleido-mastoids pull on the clavicle, the head is thrown back, and the jaw dropped forward in the effort to breathe. Profuse sweating occurs. Breathing eventually ceases in practically all cases.

It is noted that the lungs remain clear until death in many of these fatal cases. Occasionally, pulmonary edema may develop. In paralysis of the diaphragm, bronchopneumonia may occur and result in death. The heart action usually shows some changes, the rate being increased, and the rhythm affected. In other cases, the heart may remain strong and regular until the end. The fever course is about the same as that in the cases which recover.

The mental condition of these cases is interesting. Usually, the mind is clear and alert until within a few hours of death. The child concentrates his whole effort on breathing. He is nervous, excited, and requires constant attention. He fears being left alone.

The ascending type of paralysis is more common than the descending. In the latter group, the paralysis begins in the upper segment of the cord and extends downward until the lower limbs are involved.

The Bulbar Type.—In a small proportion of cases, the inflammatory process extends to the medulla and the pons resulting in the involvement of the cranial nerve nuclei. This lesion is commonly found in conjunction with spinal paralysis, but cases of bulbar involvement alone are occasionally seen. When the paralysis of the cranial nerves is isolated, it is more persistent and severe than when the involvement occurs in association with spinal paralysis. In both the bulbar and the bulbospinal paralyses, the lesions are usually unilateral.

The symptoms depend upon the nuclei involved. The *facial nerve* is most often affected, the entire nerve or one branch being disturbed. Paralysis of the *muscles of deglutition* is frequently seen; it occurs at the onset of the disease and usually lasts for a few days. In bulbar paralysis alone it may persist longer, but usually clears up after the tenth day of paralysis. Involvement of the *hypoglossal nerve* may be found in connection with disturbances of deglutition, the patient being unable to protrude his tongue. The muscles of the tongue atrophy rather rapidly, but function is not disturbed. Occasionally the *vagus nerve* is involved, resulting in disturbances of respiratory and cardiac action. Such cases often result fatally. Disturbances of *phonation* are found in the bulbospinal type; they vary from a slight hoarseness or weakness to complete aphonia. This difficulty usually occurs early and is only temporary.

Not infrequently the oculomotor nuclei are affected, giving rise to complete or partial ophthalmoplegia. Involvement of the abducens alone is the most common disturbance, resulting in convergent strabismus. This paralysis may be temporary or permanent. Blindness has occurred in some cases. Nystagmus has also been noted several times in the early part of the acute stage.

The *acoustic nerve* may be involved in some cases, and deafness result.

A symptom sometimes found associated with paralysis of the cranial nerves is ataxia. This disturbance is noted during early convalescence when the child begins to walk and has difficulty in maintaining equilibrium. The legs are slightly spastic, and the knee-jerk and ankle reflexes are increased. The condition is usually transitory and clears up within a few days. Evidently the upper neuron is affected in these cases, the lesions being located in the cerebellum, in Clarke's column, or in the pyramidal tracts as they pass through the bulb or upper cord.

The diagnosis of bulbar paralysis is evident when the cord is affected and limb paralysis is present. It is also easy to recognize this type when there are other cases of poliomyelitis in the family or neighborhood. But when the paralysis is not accompanied by a spinal involvement, or when the case is a sporadic one, the diagnosis may cause some difficulty. Paralysis of the facial nerve, or of the pharyngeal and laryngeal muscles, is particularly difficult to diagnose. If no other paralysis is present, the latter condition may be mistaken for laryngeal diphtheria. Pneumonia and meningitis may be confused with pontine cases. The history of a febrile disturbance, rigidity of the neck, and examination of the spinal fluid will help to clear up the diagnosis in questionable cases.

The prognosis seems to be good in the bulbar type of involvement. The paralysis seldom spreads to the phrenic or upper cervical centers. A fatal outcome is unusual.

THE TYPE IN WHICH THE LESION IS LOCALIZED IN THE UPPER MOTOR NEURON.

The Cerebral Type.—In this type, the chief lesion is located in the cerebrum, giving rise to paralysis of the spastic type. These cases are rarely seen, sometimes not one case occurring in epidemics of over a 1000 cases.[9] Strümpell,[10] in 1884, was the first to call attention to the cerebral type, to which he gave the name of polioencephalitis. He analyzed 24 such cases, 19 of which were under the age of four years. Medin,[11] in 1898, reported 4 cases in which he had found the cerebrum involved.

The symptoms of the cerebral type resemble those of acute meningitis. The most prominent symptoms at the onset are convulsions, vomiting, and fever; otherwise, the onset simulates that of the ordinary spinal type very closely. There may or may not be a prodromal period. The cerebral type usually shows its nature from the beginning of the attack, but in some cases, definite cerebral paralysis has not been manifested for several days or weeks. The paralysis may be hemiplegic or monoplegic. Paralysis of the face occurs less frequently and is less severe than other forms. Strabismus and ataxia also may result. The paralysis is spastic, except in those cases in which the lower motor neuron as well as the upper is involved. Examination will show exaggerated reflexes, but no reaction of degeneration.

The initial paralysis generally clears up somewhat, but there remains some loss of power and a tendency to contraction. Other sequelæ of the disease are: Signs of motor irritation, a general epilepsy or epileptiform convulsions limited to the paralyzed limb, mental and moral weakness, disturbances of speech, and athetosis of the hands. Ataxia without paralysis has been reported in some cases.

The determination of this type of cerebral paralysis as of poliomyelitic origin has been based upon the finding of such cerebral cases during the course of a typical epidemic of poliomyelitis, and upon the occurrence of both flaccid and spastic paralysis in the same patient. Experimentally, the proof of the etiology of the cerebral paralysis has not yet been determined. Transference of the virus of typical polioencephalitis to monkeys has not produced the spastic type of paralysis. Also, intracerebral inoculations have resulted in spinal, not cerebral, paralysis. Frost and Anderson[12] reported that serum from a person suffering from paralysis of the spastic type showed the same neutralizing properties as serum from a case of flaccid paralysis.

Since little study has been made of the pathological lesions of the cerebrum, their exact location is not clear. Peabody, Draper, and Dochez[7] suggest that the upper neuron may be involved in the cortex, or more commonly in the pyramidal tracts, either high up or in the cord.

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CHAPTER VII.

DIAGNOSIS IN THE PREPARALYTIC STAGE AND THE PARALYTIC STAGE.

CLINICAL DIAGNOSIS.

The recognition of poliomyelitis in its preparalytic stage is of prime importance if the spread of the infection is to be suppressed, and if appropriate treatment is to be devised in the hope of preventing the progression of the disease to the paralytic stage. Unfortunately, there is some difficulty in making an early diagnosis, as there is no definite manifestation that points out the presence of the disease. If, however, the protean symptomatology is carefully studied, and a lumbar puncture performed in a suspected case, the diagnosis may be made with a fair degree of accuracy. The possibility of making a correct diagnosis is more certain under epidemic conditions, or when the infection occurs during the late summer or early fall.

Symptoms indicating the presence of the disease are: A febrile reaction the cause of which cannot be traced; pain and stiffness in the neck; hyperesthesia; and nervous irritability. The meningeal symptoms, as demonstrated by the condition of the reflexes, Kernig's sign, Brudzinski's head and leg sign, the intracranial pressure, and the anterior spinal flexion sign, should be carefully studied.* Care should be taken to avoid error in interpreting the reflex responses. Such conditions as the failure on the part of a child to relax the muscle properly, or the tendency to assume a strained position, may account for the absence of a reaction, or for the response of the reflex on one side and lack of reaction on the other. False interpretations may easily occur in the examination of children, for they are often on the defensive while being examined. The anterior flexion sign is one of the most important diagnostic features and is among the last of the symptoms of the acute stage to disappear. This sign consists of the development of a spastic state in the posterior neck muscles, and is elicited on the attempt to flex the head.

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* A full explanation of these manifestations may be found under the Section on Symptomatology.

LABORATORY DIAGNOSIS.

In the presence of the above-mentioned symptoms, a spinal puncture is indicated.

Spinal Puncture.—*Its Value.*—Although there is a difference of opinion as to the actual diagnostic value of this procedure, since there are no pathognomonic findings in the spinal fluid that eliminate the possibility of other disease, still it is generally agreed that the changes found in the spinal fluid, if correlated with the clinical manifestations, are of value in diagnosing a case of poliomyelitis. Examination of the spinal fluid also affords the opportunity to note the presence of bacteria. If no changes are found in the fluid, poliomyelitis is ruled out, although it has not yet been determined whether a child may have the disease without changes being present in the cerebrospinal fluid. Ruhräh and Mayer,[1] in examining the spinal fluids of members of a family in which a child was ill with the disease, found no changes present, although these cases had experienced symptoms strongly indicative of poliomyelitis.

The Technique of Spinal Puncture.—There has been much discussion retarding the *position of the patient.* Some writers advise the sitting posture; others disapprove of it because of the danger of breaking the needle. The recumbent position with the patient lying on his side is preferable to any other, since the attendant can control him more easily in this position. It is understood that a right-handed operator can work more conveniently with the patient lying on his right side.

Regardless of the position advocated, it is important to flex the spine sufficiently for the anatomical structures to be recognized. The legs should be drawn up until the knees touch the abdomen, and the head and shoulders should be bent forward on the chest. This position produces the separation of the vertebræ posteriorly, and makes the ligaments tense.

It is very important to keep the child perfectly still. One attendant can usually hold an infant quiet, but in the case of an older child, more assistance may be necessary. If only one attendant is available, he can keep the patient in good position either by standing back to the patient and putting one arm around his neck and the other around his knees, or by passing one arm between the patient's thighs and bending his head so that he can grip the back of the neck, at the same time flexing the head on the chest with the other hand (Youland,[2] Lott[3]). Some writers recommend securing a nervous patient by means of a sheet twisted in rope form. Others suggest allowing the patient to sip water during the operation.

Anesthesia, either general or local, is seldom necessary. The operation is over in a few seconds, and the patient is rarely so restless that he cannot be controlled. The pain is very transient, being similar to that caused by the prick of a pin. Occasionally, a general anesthetic is required if the patient is delirious or unmanageable. In adults the use of an anesthetic often may be avoided by a hypodermic injection of morphine sulphate, gr. $\frac{1}{4}$ and atropin sulphate, gr. $\frac{1}{20}$, two hours before the operation.

Equipment.—The type of needle used for rachicentesis is very important, and a subject that has received much attention. Various needles have been devised and used, but the outstanding requisites of all have been in the main: Exquisite sharpness to avoid unnecessary pain, flexibility, and the attachment of a steel trocar, ground so as to flush with the cutting edge of the needle. No. 18-gauge needle is the proper size. Bluemel[4] recently called attention to a small Luer needle with a sliding sleeve, which serves as a holder and prevents bending. By reducing the size of the hole made in the dura, this needle helps to avoid puncture headache. It is difficult to establish an exact standard for the proper length of the needle. Levinson,[5] who studied the needles used in several hundred spinal punctures performed on children, found that the length of the needle used in children up to the age of twelve years varied from 2 cm. to 4 cm., and in patients over sixteen years of age from 4.1 cm. to 10 cm. The appropriate length of the needles for adults would seem to vary from 7 to 10 cm., and for children, from 5 to 8 cm.

The other necessary articles for performing a spinal puncture are: A 20-cc. Luer syringe, a glass funnel and proper tubing attachment to fit the needles, several sterile test tubes, sterile towels, and a pus basin.

Asepsis.—The same precautions should be taken as for other operations. The operator should scrub his hands carefully, and the needles and tubes should be boiled thoroughly. The lumbar region is first cleansed with alcohol, then dried, and painted with a tincture of iodine.

Location and Route of Puncture.—The usual site for puncture is between the third and fourth lumbar vertebræ, but occasionally the interspace between the second and third lumbar vertebræ may be used. The level for the site of puncture is found by drawing a line from the upper part of the iliac crests through the fourth lumbar vertebra and selecting a point just above the line.

There is some diversity of opinion as to the route of puncture. Many writers advocate the median route in children and the lateral route in adults. In children, the needle can easily be introduced in the middle line, for when the spine is flexed there are fairly wide spaces between the spinous processes. Even in infants there is still ample room for the passage of the needle. In older children and adults, however, many operators believe that the interspinous ligament blocks the entrance of the needle when it is inserted in the mid-line, and for this reason they recommend the lateral route.

A few operators, including Regan,[6] Sophian,[7] Neal and Dubois,[8] and Lorenz,[9] prefer the median route in adults. Lorenz favors it strongly, as he obtained dry taps in a number of cases in which he used the lateral route, while with the median course he obtained fluid in all but 2 per cent of his cases. Regan made an investigation to determine just what resistance the interspinous ligament offered. As a result of his clinical and autopsic study of a series of adults, he found that the interspinous ligament is an aid rather than a hindrance to the insertion of the needle. This ligament is composed of two folds of layers of lateral fibers, with a distinct line of cleavage between them. It is attached along the whole length of the lower border of each spinous process from its root to its summit, and extends downward to the same part of the superior border of the spinous process below. When the needle enters in the middle line, it first pierces the supraspinous ligament and then passes along between the layers of the interspinous ligament to the interarcual space. The interspinous ligament was found to be an aid even in the case of muscular individuals, several of whom were operated successfully by the median route.

Direction of the Insertion of the Needle. In children, most authorities recommend a slightly upward inclination of the needle, in order that it will pass between the spines of the vertebræ. Regan and a few other operators have found that the needle may be introduced perpendicularly to the spine, without its meeting any obstruction. Those writers advocating the lateral route in adults claim the needle should be directed forward, upward, and slightly inward toward the mid-line. Regan found that, in spite of the distinct downward inclination of the lumbar spinous processes in the adult spine, the intervals were wide enough, if the spine was well bowed, to allow the passage of the needle in a perpendicular direction. If introduction by this course is found impossible, the needle should be directed obliquely upward at an angle of 60 to 45 degrees.

The median route and the perpendicular insertion of the needle have certain advantages over the lateral route and the upward inclination of the needle. The technique is easier to learn; when the needle goes straight in, the exact angle at which it should be inclined is known. The possibility of wounding the periosteum, or encountering bone and perhaps breaking a needle, is less. There is little danger of passing beyond the limits of the interarcual space, or injuring a nerve filament or spinal bloodvessel. Dry taps are uncommon when the median route is used.

The depth to which the needle must be inserted before the fluid is reached varies with the age, development, and adiposity of the patient. In adults, the needle is usually introduced for 6 to 7 cm. before it penetrates the dural sac; in children the distance is much shorter. A slight resistance is felt when the needle has reached the dura. When this resistance gives way suddenly to lack of pressure, the fluid has been reached. The trocar is then withdrawn, and the fluid runs out. It may drip slowly or flow rapidly, depending upon the pressure. If an obstruction should be encountered while introducing the needle, it is best to withdraw it slightly and insert it in a different direction.

The Amount of Fluid Withdrawn.—For diagnostic purposes, 5 to 10 cc. of fluid are usually withdrawn. If the puncture is performed as a therapeutic measure to relieve pressure, the fluid may be allowed to escape until only an occasional drop appears. After the fluid has been withdrawn, the needle is removed quickly, and the puncture area washed off with iodine and alcohol. Flexible collodion, or a piece of sterile gauze fastened with a piece of adhesive plaster is then applied. The patient should lie flat on his back in bed for twenty-four hours. During this time he should have a liquid diet.

Possible Difficulties Encountered in Puncture.—At times, the flow of fluid may be either restricted or too rapid. In case of the former condition, a slight twitch of the needle will usually cause the flow to increase. In some cases, the restricted flow may be due to introducing the needle on a slant, causing it to come in contact with the lateral margin of the dural space. In such conditions it is well to withdraw the needle and redirect it straight. A rapid flow of fluid may be checked by placing the finger over the lumen of the needle.

Occasionally it may happen that a dry puncture is obtained, either because of peculiar anatomical conditions, or because of certain pathological lesions that prevent the needle from reaching the lumbar part of the spinal canal. In such cases another puncture should be performed.

Another difficulty one often meets is the presence of blood in the specimen, due to the puncturing of a vein. A loosely fitting trocar is responsible for this trouble. A little blood does not cause macroscopic changes, but the amount need not be very large to produce a yellowish shimmer and an opalescence of the fluid. The presence of blood interferes with an accurate chemical investigation. When blood is seen to appear, the needle should be withdrawn slowly for a little way. If the blood still continues to flow, a fresh puncture should be made at a different site.

After-symptoms of Spinal Puncture.—Lumbar puncture is often followed by headache, accompanied at times by nausea or vomiting, dizziness, and a feeling of faintness. It rarely is experienced before the day following the puncture, but there have been cases in which it began either immediately after rachicentesis or as late as three days after the procedure. Its occurrence depends upon the activity of the patient and the condition of the circulation of the cerebrospinal fluid. The pain is a throbbing, rather severe one, usually in the frontal region, but occasionally in the occipital section. The pain is present only when the patient sits up, and it is greatly increased by activity. The duration of the pain varies; it has cleared up in five or six days in some cases, but in others it has lasted intermittently for as long as two weeks. Headache can be prevented by keeping the patient flat on his back for a few days following operation. When headache occurs, recumbency is the only means of relief.

Various explanations of the cause of this headache have been advanced. Among those considered and proved as unimportant are: The amount of fluid withdrawn; the position of the patient during the operation; the rapidity of the flow; the degree of intraspinal pressure; and the need of replacing the serum withdrawn by autogenous serum. It is possible that the oozing of the blood into the spinal canal may have some influence in causing headache, as Baar[10] found that 13 out of 18 cases, in which bloody fluid was collected, developed headache which lasted from two to nineteen days. The explanation advanced by MacRoberts,[11] which is concerned with the closure of the hole punctured in the dura membrane, seems most feasible. Briefly, it is as follows: When the needle punctures the hard dura, it leaves a clean-edged hole. Under normal conditions, this hole is blocked when the arachnoid tissue, dropping from the point of the needle, is brought closely against the dura mater by the pressure of the fluid within. But, supposing the arachnoid tissue, which is delicate, clings to the needle and is pulled through the dural hole, might it not form a sort of wick for the drainage of the spinal fluid. The constant leakage of fluid from the cerebrospinal sac into the epidural space, during the week or so following spinal puncture, destroys the fluid support of the brain, and allows forcible descent of the pons on the clivis of the occipital bone, so that prolapse of the medulla through the foramen magnum occurs. The basilar veins on the clivis of the occipital bone depend upon the cushion of the cerebrospinal fluid to keep pressure off the pons. When the patient sits up, the cushion of fluid is absent, and the weight of a part of the brain is imparted through the pons to this communicating basilar plexus. The blood leaving the skull is forced back and is compelled to flow through congested routes, causing a rise of venous pressure. When the patient lies down, the pressure falls, and the weight is removed from the veins on the clivis, with the result that headache ceases.

Complications of Spinal Puncture.—Although it is the opinion of a large group of clinicians who have had extensive experience with lumbar punctures, that the procedure is simple and safe if properly carried out, there are a few complications to be avoided.

There may sometimes be a slight injury to the meninges and the nervous parenchyma, which causes a number of investigators to harbor fears of the possibility of infection of the meninges, and to urge a conservative use of rachicentesis. Much of the apprehension is due to the observations of Flexner and Amoss,[12] who demonstrated that even a slight hemorrhage from puncture allowed the entrance of the poliomyelitic virus from the blood into the central nervous system. Webster,[13] working with monkeys, showed that lumbar puncture may facilitate the infection of the meninges by impairing the integrity of the choroid plexus and the meningeal lymphatics, and allowing the extravasation of the blood, which may be infected, into the cerebrospinal space.

The breaking of a needle, an accident that occasionally occurs, may be avoided by using one that is not too long or pointed, and by keeping it rigid during introduction. In case of accident, it is best to deter the removal of the needle until after the disease has run its course.

The Cerebrospinal Fluid.—*The Normal Fluid.*—A knowledge of the nature of the normal cerebrospinal fluid is essential, if the significance of the pathological changes that take place in infections of the nervous system is to be appreciated. The normal fluid is clear, transparent, and to the naked eye is not distinguishable from water. The pressure with which it escapes varies according to the position of the patient and the caliber of the needle, but it usually flows drop by drop, at about 10 to 15 drops per minute. Its specific gravity varies from 1.003 to 1.013, but ordinarily it is about 1.007 or 1.008.

Spinal fluids are subjected to three types of examination: Cytological, bacteriological, and chemical. The constituents of normal fluid are: Water, fat, cholesterin, chlorides, sulphates, phosphates, alcoholic extracts; traces of albumin, protein salts, sugar, cholin, albumoses, urea; serum-albumin, and serum-globulin. The fluid is alkaline in reaction. As regards the cytology of the normal fluid, microscopic examination reveals only a few endothelial cells and a few lymphocytes. The number varies from 2 to 10 per cmm., the average being about 8 cells per cmm. No bacteria are found in the fluid.

THE CEREBROSPINAL FLUID IN POLIOMYELITIS.—*Macroscopic Appearance.*—When the fluid is first withdrawn, it is usually clear and flows under increased pressure; in only a small number of cases is it cloudy or hazy. The presence of a yellowish tinge signifies that the fluid contains blood. There is often a thin filament of fibrin on the surface of the fluid, but since such a formation appears in many pathological fluids of sources other than the poliomyelitic virus, little significance can be attributed to it.

Two other types of spinal fluid are met with occasionally in poliomyelitis. They are not peculiar to this disease alone, and may be found in other infections. The first one, to which Neal[14] and Mammack[15] called attention, presents the "syndrome of Froin," consisting of a clear yellow color, rapid coagulation of the fluid, and the presence of large amounts of globulin and lymphocytes. The second rare type is seen when the hemorrhagic process has been unusually extensive. The fluid shows red blood cells evenly distributed throughout. When such fluid is being collected in the various test-tubes, it will be noted that all the specimens are homogeneous, whereas if the fluid is bloody from an accidental puncture of a vein, only the first tube will show signs of the hemorrhagic invasion.

Zingher[16] has called attention to the ground-glass appearance of the fluid when viewed by transmitted light in a dark room. This is due to the increased number of lymphocytes, which may be seen with the naked eye or better still with the aid of a magnifying glass. The cells can be set in motion by shaking the test-tube gently. If the fluid is left standing for a number of hours, the particles settle to the bottom of the tube, and the fluid becomes clear. The ground-glass appearance is again reproduced by shaking the tube. Two precautions must be taken in making this test. First, a microscopic examination must be made, to avoid mistaking the ground-glass appearance due to the increased lymphocytes for that due to the presence of a few red blood cells which may produce a similar opalescence; second, it must be borne in mind that this ground-glass appearance may be present in other infections, especially in tubercular meningitis and in early purulent meningitis with slight cellular reaction.

Cytology.—One of the most important tests for diagnostic purposes is the determination of the number and character of the cells in the spinal fluid. As a rule, there is a definite increase in the number of cells. The amount of the increase varies in different cases and at different stages of the disease. The normal fluid contains from 5 to 10 cells per cmm.; the fluid in poliomyelitis may contain from 16 or 20 to hundreds or even thousands per cubic millimeter above normal. The cell-count is usually highest during the first week, after which it decreases, until at the end of the second week the condition is practically normal. Draper[17] noted exceptional cases in which there was no increase in the number of cells until several days after the onset of the disease.

Draper sought to establish a basis for prognosis from a study of the relationship between the increase in the number of cells and the final outcome of the disease. Although he could not definitely determine that paralysis did not develop in cases with low cell-counts, or that it occurred in cases with high cell-counts, still he found that those having a cell-count below 100 per cmm., within the first twelve hours, were less likely to develop paralysis than those with cell-counts of 500 or more. Draper considered the count of little prognostic value after thirty-six hours from the onset of the disease. Other writers believe the cell-count during the phase of meningeal involvement does not furnish a basis for the prognosis.

The predominating cell in the early stages before paralysis occurs is similar to the polymorphonuclear. Menninger[18] believes these cells may be wandering tissue cells, or clasmatocytes of Marchand. In some cases this type of cell may prevail to the extent of comprising from 80 to 90 per cent of the total number of cells present.

After meningeal invasion, the predominance of the polymorphonuclears gives way to cells of the mononuclear type, chiefly of the lymphocytic variety. In a few cloudy fluids, Neal[14] found that over 50 per cent of the cells were polymorphonuclears, but these cases are exceptional. Neal also called attention to the presence of some large mononuclear cells, which appear frequently in fluids of poliomyelitic cases, and which seem to be endotheloid in character. Polyform cells are also present. It may be difficult to classify the cells in some cases on account of their degenerated condition.

(*a*) *The Protein Content.*—Both the globulin and albumin are usually slightly to moderately increased. The excess of globulin is comparatively slight during the early phase of the disease, but the content rises gradually until the third week, when it is most marked. As a rule, it then begins to diminish, although it may persist long after the acute symptoms have passed. The increase in albumin runs a similar course, reaching its maximum in the first few weeks, and then decreasing, or persisting late. This gradual rise in the protein content is in contrast to the cell-count, which tends to be high in the early part of the disease and to diminish gradually.

(*b*) *The Foam Test.*—This test is immediately dependent upon the increased protein content of the fluid. It is carried out by shaking a test-tube half full of fluid. This shaking creates a foam, which persists for one-half hour or longer. It is true that a foam is produced when any spinal fluid is shaken, but that produced in the fluid of a poliomyelitic case is more dense, greater in volume, and more persistent. This test is sometimes of value in helping to make a diagnosis before laboratory facilities are obtainable for further examination.

(*c*) *Lange's Colloidal-gold Test.*—This test has been tried by many investigators, and they have found it very valuable in helping to recognize poliomyelitis. Its results are more constant and definite than the findings of either the cell or globulin increase. When the test is correlated with the clinical symptoms and other laboratory data, it is very helpful in making the differential diagnosis between poliomyelitis and the various forms of meningitis.

The purpose of the test is to estimate the presence of protein in the cerebrospinal fluid. This is determined by adding a preparation of colloidal gold to ten dilutions of spinal fluid, ranging from 1 to 10 to 1 to 5120. The results are interpreted according to the change in color and classified in zones as follows: Zone 1 (paretic zone), maximum precipitation from 1 to 10 to 1 to 160, with complete decolorization; zone 2 (luetic), maximum precipitation from 1 to 40 to 1 to 160, with decolorization up to 4 (light blue); zone 3 (meningitic), maximum precipitation beyond 1 to 160, producing a maximum decolorization of 3 (blue).

Felton and Maxcy,[19] and Regan, Litvak, and Regan,[20] have made studies of this test and published their results. The fluids analyzed were obtained at various intervals from the fourth to the one hundred and twenty-third day of the disease. The experimenters found that there was always a reaction with colloidal gold in all poliomyelitic fluids examined during the acute phase of the disease. The reduction occurred consistently in the zone corresponding to a luetic spinal fluid. The average reaction in the first and second weeks of the disease showed very similar characteristics, consisting of a gradual increase in reduction in the higher dilutions of the spinal fluid, beginning in the reddish-blue zone in 1 to 10 dilutions, and extending into the lilac zone in 1 to 40 to 1 to 80 dilutions. A gradual decline of the curve was then noted. The decrease was very gradual, however, and a normal reaction was never encountered before the end of the third week. Most cases required a much longer period for the reaction to become normal. Regan,[21] in a study of a series of 74 fluids, found that in 70 per cent the curve was still elevated at the ninth week. As a rule, such cases showed considerable residual paralysis with some polyneuritis, or slight paralysis with marked polyneuritis. From such findings, many writers have come to believe that there is a relationship between the duration of the positive colloidal-gold curve and the acute inflammatory stage of poliomyelitis. Other points of interest were noted in connection with this test. Regan, Litvak, and Regan found that no characteristic reaction was obtained in fatal cases; also that the positive reaction bore no constant relation to the presence or to the disappearance of globulin. Neither was there a close relation between the cell-count per cubic millimeter and the colloidal-gold reaction, although the curve of the latter subsided with the decrease in the number of cells. This decline was very slow, however, so that the colloidal-gold reaction remained elevated long after the cell-count was normal.

(*d*) *The Reduction of Fehling's Solution.*—A prompt reduction of Fehling's solution is obtained in the majority of cases. The fluid containing an excess of protein usually shows a poorer reduction. The diagnostic value of the reduction lies in making a differential diagnosis between poliomyelitis and meningeal inflammation, as in meningitis there is usually no reduction of Fehling's solution.

(*e*) *Chemistry.*—Chemical studies of the variations in the protein, nitrogen, urea, ammonia, creatin nitrogen, creatinin, and sugar content have so far revealed nothing of diagnostic value.

The Technique of Examination of the Spinal Fluid.—*The Differential Count.*—Two or 3 cc. of spinal fluid, whether clear or cloudy, are centrifuged in a test-tube for forty minutes to one hour at high speed. The supernatant fluid is drained off, and the sediment in the bottom of the tube taken up with a capillary pipet. The cells in this sediment are thoroughly mixed, and smears made. After the smears have dried, they are passed through the flame to fix them, and stained. For staining purposes, Wilson's, Jenner's, Giemsa's, or Hasting's method may be used. The increase in cells is then estimated and the presence of mononuclear, polymorphonuclear, endotheloid, and polyform cells noted. It is impossible to make an accurate cell-count, but at least 300 cells should be counted to make the estimate at all reliable (Ruhräh and Mayer[1]).

The Total Count.—In estimating the total cell-count of the spinal fluid, the same technique is used as in calculating the total white count of the blood. The Fuchs-Rosenthal counter is generally preferred. A staining fluid is made up of methyl violet, 0.1; glacial acetic acid, 2; water, 50. A special counting chamber is designed for counting the cells of the spinal fluid. The examiner, using a white cell pipet, draws the staining fluid to mark I, and the spinal fluid to mark XI. All the cells in the entire ruled area are counted; the total number divided by three gives the number of cells per cubic millimeter (Ruhräh and Mayer[1]).

Chemical Tests.—(*a*) *Tests for Globulin.*—Some writers prefer Pandy's carbolic test, others prefer the Noguchi butyric acid test, and still others use Nonne's method, or the ammonium sulphate test of Ross and Jones. Of the three, Pandy's method gives a more marked reaction and for this reason is considered so delicate that some experimenters believe it may lead to questionable results in the case of normal fluids. However, many other laboratory workers have found Pandy's test simple, easy, and reliable. In brief, it consists of mixing 80 to 100 cc. of pure carbolic acid with 100 parts of distilled water, shaking, and keeping the mixture at room temperature for three to four days, with frequent shaking. The top watery fluid is poured off and used as the reagent. One or more cubic centimeters of the fluid are poured into a test-tube, and 1 drop of the spinal fluid added. If no cloudy precipitate forms within five seconds, the reaction is negative. Under normal conditions no change takes place. If, however, the protein content of the spinal fluid is increased, a bluish-white cloud is seen at the point of contact. After a short time this ring of smoke settles to the bottom. The intensity of the reaction is estimated from the density of the bluish-white cloud, and is found to vary according to the amount of globulin present (Ruhräh and Mayer[1]).

(*b*) *Test for Albumin.*—The following tests may be used: The nitric acid test, the heat and acetic acid test, the Kjeldahl method, and Tsuchiya's modification of Esbach's method. The total protein content may be estimated by the Kjeldahl method or the Tsuchiya method. Tsuchiya's test is as follows: The reagent consists of phosphotungstic acid, 1.5 gm.; concentrated hydrochloric acid, 5 cc.; and ethyl alcohol, 95 cc. Special albuminometer tubes are employed, which bear two marks, one *U*, indicating the point to which the urine must be added, and the other *R*, the point to which the reagent is added. The lower portion of the tube up to *U* bears a scale which reads from 1 to 7, corresponding to the amount of albumin per 1000. The tube is filled to *U* with the spinal fluid, and the reagent is then added to the point *R*. The tube is closed with a stopper, inverted twelve times; and then set aside for twenty-four hours. At the expiration of that time the amount per 1000 in grams can be read (Ruhräh and Mayer[1]).

(*c*) *Test for Glucose.*—The presence of glucose is tested by using an equal amount of Fehling's solution and spinal fluid.

Other Diagnostic Features.—*The Blood Examination.*—La Fetra,[22] Müller,[23] Gay and Lucas,[24] Peabody, Draper, and Dochez,[25] and Neal[26] have made the most extensive observations on the condition of the blood in poliomyelitis. The results of their investigations are somewhat conflicting. Müller reported that in 15 cases he found a tendency toward a leukopenia, with a slight increase of lymphocytes in the febrile stage. On the other hand, Peabody, Draper, and Dochez, in the examination of 59 cases, found a constant leukocytosis, a constant increase of polymorphonuclears, and a diminution of lymphocytes from 15 to 20 per cent. La Fetra also found a leukocytosis. The duration of the abnormal condition of the blood varies in different patients, but the blood picture never returns to normal before the third week of the attack.

Peabody, Draper, and Dochez believe that although the blood picture yields no specific feature peculiar to poliomyelitis, the finding of a leukocytosis of 15,000 to 30,000 and an increase of polymorphonuclear cells at the expense of the lymphocytes are of help in making a diagnosis, if this data is correlated with the clinical and spinal fluid examinations.

The Neutralization Test.—The value of this test is questionable, and it is impractical for ordinary diagnostic purposes. It consists of taking the serum from a suspected case of poliomyelitis and incubating it with an active virus. The mixture is then injected intracerebrally into monkeys. At the same time the virus alone is inoculated into a control. If the control monkey develops the disease, and the other monkey suffers no ill effect, it is concluded that the virus has been neutralized by antibodies in the serum. There are certain objections to this test. The difficulty of securing the large number of animals that would be required for such tests, and the length of time necessary for the elaboration of the experiment render this method of little practical value. It is also true that the serum of persons who apparently have not had a recent attack of the disease has been found to neutralize the virus.

The Complement-fixation Test.—Attempts to find a specific antigen for complement fixation have so far been unsuccessful.

Urine Examination.—Neal[26] found no changes in the urine which were of diagnostic value.

DIAGNOSIS IN THE PARALYTIC STAGE.

In many cases the paralysis is so apparent that diagnosis presents little difficulty. For instance, complete flaccid paralysis of a whole limb is perfectly obvious. In an older child or adult, the loss of power may often be determined by simply watching the patient or asking him to move his extremities, or if pain and tenderness permit, by gently manipulating the involved parts.

There are other cases, particularly among young children, that require very careful examination to determine the exact paralytic condition. Sometimes pain, not weakness, may be responsible for the failure to move a limb. An isolated paralyzed muscle or group of muscles may be easily overlooked. Evidence of paralysis of the gluteal muscles or the quadriceps may not be noted until the child collapses upon standing. The sudden stretching of a muscle, as by straightening a flexed forearm, will reveal the lack of muscle tone by the failure of the flexor group to resist. In some cases it may be necessary to prick the different areas with a pin to determine whether or not the child has muscle control of the extremity. In determining the paralysis in infants, it is of help to hold the child in different positions and note the control of the extremities.

The condition of the reflexes, which may be diminished or lacking entirely, helps to throw some light on the diagnosis. In the late paralytic stage, atrophy and retarded growth, due to the impairment of the circulation, are other significant signs.

A detailed account of the examination of the muscles will be found under the sections treating paralysis of the various extremities. There are several important precautions to be taken in examination of the muscles, which it might be well to repeat at this time. The muscle must be put in such a position that it works against the force of gravity. If not, the child instinctively will utilize the weight of the limb to make the movement that the paralyzed muscle cannot perform. The examiner must also make sure that the child is not accomplishing a movement by the substitution of the power of muscles that are not regularly involved in the movement. Children often circumvent a weakness in this way.

Electricity is of some value in examining a paralyzed muscle. A reaction of prognostic importance, which may be obtained by means of both the faradic and galvanic currents, is the R.D.—the reaction of degeneration. This reaction appears toward the end of the first week, or at the beginning of the second week. If there is no response to the faradic current by the end of the second week, it is usually assumed that the nerve and muscle have degenerated. If there is some faradic excitability present, only a partial degeneration has occurred, and the muscle may recover. It must be borne in mind that lack of response to faradism does not always indicate that the muscle will never regain its power, for in some cases the voluntary power is recovered before the faradic excitability. Changes in response to the galvanic current are of serious import; if the muscle fibers show a complete reaction of degeneration with a sluggish muscular contraction, there is little hope of return of voluntary motor power or functions.

Electricity should be used only by one who thoroughly understands its application. It requires skill and practice to make an accurate electrical diagnosis. In the case of the young child, who is likely to be frightened by the electrical apparatus, a great deal of tact must be used to keep him quiet.

DIFFERENTIAL DIAGNOSIS.

Poliomyelitis in both its early and late phases may be confused with several other diseases, which apparently run a similar course, or which show a weakness simulating the paralysis that occurs in poliomyelitis. For the discrimination of such cases, a record of the history of the onset is of greatest importance and should be studied thoroughly. In addition, a careful comparison of the clinical symptoms of poliomyelitis with those of the confusing disease; a thorough study of the conditions under which the disease occurred; an examination of the weakened limb, with observation of the condition of the reflexes; and finally, examination of the spinal fluid, are all means that aid in making a correct diagnosis. In some instances the appearance of the cerebrospinal fluid obtained in a case of poliomyelitis is so similar to that of other meningeal infections, that a differentiation of the fluids is necessary to determine the exisiting condition.

The diseases with which poliomyelitis may be confused are conveniently grouped under four heads:

1. Acute infectious diseases without paralysis.
2. Diseases involving the nervous system.
3. Pseudoparalysis or spasm.
4. Conditions presenting loss of power due to lesions other than poliomyelitis.

Acute Infectious Diseases Without Paralysis.— *Typhoid.* —This malady presents early symptoms of extreme prostration, high temperature, delirium, tremor, and headache —manifestations that might be confused with those of a case of poliomyelitis. The insidious onset of typhoid, the continuous fever, rash, a low white count, positive Widal reaction, and finally the absence of paralysis exclude the possibility of infantile paralysis.

Rheumatism.—Extreme hyperesthesia and fever, sometimes present in poliomyelitis, may lead to the mistaken diagnosis of rheumatism. Acute pain, swelling, and redness about the joints, as well as a history of previous attacks, will assist in making the diagnosis of rheumatism. The writer recalls a case in which the severe muscle and skin tenderness led to the incorrect diagnosis of rheumatism. The child had a bilious attack, during which the temperature registered 102 to 103° F. His muscles were very sensitive and he suffered severe pain. In his attempt to secure relief from the symptoms, he held his limbs in deformed positions. The pain and sensitiveness continued for a period of eight to nine weeks, and the patient could not be moved. A diagnosis of rheumatism had been made, but the condition was due to a severe attack of poliomyelitis, characterized by an unusual amount of muscle and skin tenderness, which had persisted for a much longer period than in the average case. When the joints had been placed at rest in proper position, and the deformities corrected, the muscle tenderness and sensitiveness disappeared very quickly.

Diseases of the Respiratory Tract. —Respiratory diseases, such as *influenza,* are not likely to be confused with poliomyelitis, as they usually occur during the winter and spring months, and affect adults much more frequently than children. Furthermore, catarrhal symptoms are absent in poliomyelitis.

In very exceptional cases of poliomyelitis, the tonsils may be affected to such an extent that their inflamed condition leads to the incorrect diagnosis of *tonsillitis.* In the latter disease, however, there is usually a history of previous attacks, and possibly a rheumatic tendency.

Paralytic involvement of the laryngeal muscles may lead to the suspicion of *croup, laryngitis,* or *laryngeal diphtheria.* The presence of paralysis elsewhere and the absence of signs of diphtheria serve as a means of differentiation.

Paralysis of the respiratory muscles, which gives rise to rapid respiration, may suggest *pneumonia.* Examination reveals a paralysis of the thoracic muscles or of the diaphragm without involvement of the lungs.

Gastro-enteritis.—This infection, like infantile paralysis, occurs in the summer months and early autumn. Its slow onset, gradual rise in temperature, and the absence of drowsiness help to differentiate it from poliomyelitis.

Diarrhea.—This disease is often accompanied by meningeal symptoms, which may lead to some confusion in making a diagnosis. A lumbar puncture is a certain means of differentiating diarrhea from poliomyelitis.

Acidosis.—The loss of power and the nervous symptoms sometimes accompanying an acidosis may cause some difficulty. The diagnosis can be made from the sweet odor of the breath and the diacetic reaction in the urine. Lumbar puncture can always be resorted to if necessary.

Nephritis with Uremia.—The coma and convulsions in uremia may be confusing, but examination of the urine settles any doubt. In some cases, a lumbar puncture may be necessary.

Diseases Involving the Nervous System.—Diseases involving the nervous system may be differentiated from poliomyelitis by a careful study of the clinical symptoms and history of the onset; other diseases, which present clinical symptoms very similar to those of poliomyelitis, can be diagnosed only by differentiating between the spinal fluid of the given case and the fluid of a poliomyelitic case.

Among the diseases that may be distinguished by the clinical symptoms are the following:

Lethargic Encephalitis.—The prodromal period of this infection, presenting symptoms of fatigue, headache, and lethargy, suggests a relation between this disease and poliomyelitis. It is not uncommon to find both infections in the same patient. Poliomyelitis, however, is essentially a disease of the spinal cord, and the cerebrum is seldom attacked. Lethargic encephalitis, on the other hand, presents primarily cerebral signs, and in only exceptional cases is there spinal cord involvement. Poliomyelitis occurs mainly in children; it is a disease peculiar to the summer months, and its onset is acute. Lethargic encephalitis attacks all ages indiscriminately; it is more prevalent in winter; and its onset varies in duration. The presence of an epidemic aids in making a diagnosis. The findings in the spinal fluid do not seem to be of much value.

Cerebral Thrombosis, Cerebral Abscesses, and Tumors of the Brain and Cord.—The general symptoms of drowsiness, irritation, convulsions, and paralysis presented by cerebral thrombosis may suggest poliomyelitis. The local signs, consisting of cyanosis of the face, marked edema of the eyelids and face, and protrusion of the eye, help in differentiation.

The slow development of tumors and abscesses of the brain, the mental condition, and the local symptoms in these cerebral lesions make the diagnosis clear.

Acute Transverse Myelitis.—This disease presents symptoms of pain, hyperesthesia, motor disturbances, and paralysis of the sphincters; if the lumbar region is involved, there is an increase in the reflex response. Bed sores develop early in the course of the disease. At a later period the gait is spastic, the muscles atrophy slowly, and a paralysis develops which does not improve. While some of the symptoms may be confused with those of poliomyelitis, a consideration of the general symptomatology outlined above prevents any error in diagnosis.

Progressive Muscular Atrophy and Hypertrophy.—Progressive muscular atrophy is characterized by a slow onset without any loss of reflexes. It rarely occurs in childhood.

The progressive muscular hypertrophy is likewise of slow onset, and the increase in muscular tissue, especially in the calf, is marked.

Peripheral Neuritis. The polyneuritic form of poliomyelitis simulates peripheral neuritis very closely. The latter, however, is not essentially a disease of childhood; when in exceptional cases children are affected, the neuritis follows an acute specific fever, such as diphtheria. The onset of peripheral neuritis is more gradual than that of poliomyelitis. Paralysis occurs later in the disease than is customary in infantile paralysis, and the maximum stage is reached more slowly. The palate and fauces and the eye muscles of accommodation are usually affected. The electrical reactions remain normal. There is pain on pressure of the nerve trunks and the atrophy is less pronounced. A normal fluid is usually found in postdiphtheritic paralysis. The history of the onset, the clinical symptoms, the spinal fluid findings, and the course of the paralysis will aid in distinguishing the postdiphtheritic paralysis from the polyneuritic form of infantile paralysis.

Chorea.—A careful examination should eliminate any difficulty in differentiating this disease from poliomyelitis.

Among the diseases presenting spinal fluid findings that must be differentiated from those of the cerebrospinal fluid of poliomyelitis are the following: Tubercular meningitis; syphilis of the central nervous system, especially acute syphilitic meningitis; meningism; and occasionally an early case of purulent meningitis. All the fluids are practically clear, resembling that of poliomyelitis. Early cases of purulent meningitis and occasionally cases of tubercular meningitis may show cloudy spinal fluids similar to the turbid fluids sometimes found in poliomyelitis.

Tubercular Meningitis.—The prodromal symptoms of this malady are drowsiness, vomiting, irregular respiration, irritability, fever, slight gastro-intestinal disturbances, and rigidity of the muscles, all of which symptoms may be found in cases of poliomyelitis. The general appearance of the patient, the irregular temperature curve, and the duration of the prodromal period help in differentiating the disease from infantile paralysis. As a rule, a spinal puncture is necessary for a final decision. Rachicentesis, however, presents a fluid very similar to the poliomyelitic fluid. It is usually clear, with an increase in the mononuclear type of cell. If tubercle bacilli are present, a positive diagnosis may be made, but if absent, the diagnosis is uncertain. In such difficult cases the following signs are of help: The reduction of Fehling's solution is usually delayed in tubercular meningitis, whereas in poliomyelitis the reaction is nearly always immediate; in the tubercular meningitic fluid there are usually more cells per cubic millimeter, and the increase in albumin and globulin is more marked. Regan[21] found that the colloidal-gold test helped in differentiation. Youland[2] noted that the sugar content was decreased or absent in over 50 per cent of the fluids from cases of tubercular meningitis, but that it was not affected in poliomyelitis, as determined by qualitative tests. In very rare cases, when both clinical and laboratory investigations yield indefinite results, animal inoculation is necessary to determine the character of the disease.

Acute Syphilitic Meningitis.—This disease may simulate acute poliomyelitis very closely, both in clinical signs and in spinal fluid findings. A positive Wassermann reaction renders the diagnosis clear in these cases. The colloidal-gold reaction test and the protein test also aid in diagnosis. If the case is one of poliomyelitis in an old syphilitic condition, the clinical symptoms and progress of the disease help in making the diagnosis. Clinical symptoms that assist in differentiating the diseases are: The gradual onset of acute syphilitic meningitis, lasting from a month to six weeks, during which period the patient suffers an optic neuritis, is nervous, has vomiting attacks without nausea, and sometimes ataxia; convulsions; and paralysis of the extrinsic ocular nerves.

Meningism.—Evidences of meningeal involvement, such as drowsiness, retraction of the head, convulsions, and vomiting, may give rise to some difficulty in diagnosis. Examination of the fluid is helpful. As a rule, the fluid in meningism is clear, increased in quantity, and nearly always of normal character. Neal[26] found that the few exceptions of increased cellular and protein content were in fluids of cases with severe convulsions, in cases of severe whooping-cough, or in meningitic fluids removed just before death. Lange's colloidal-gold test seems to be of great help in differentiation. Neal and Dubois reported that in several cases of meningism in which a slight increase in globulin and albumin was found, the Lange test gave a negative reaction.

Purulent Meningitis.—All forms of purulent meningitis present a history of sudden onset, and general symptoms of temperature, headache, irritability, delirium, and gastro-intestinal disturbances. Of course, a history of pneumonia, or an operation on the nose or throat, suggests that this meningitis is of pneumococcic or streptococcic origin, but nearly all cases can be definitely determined by examination of the spinal fluid.

In the early stages of purulent meningitis, the slightly turbid fluid is very similar to the cloudy fluid sometimes found in poliomyelitis. It is only in rare cases that a clear fluid is found in purulent meningitis within the first twenty-four hours of the disease. The fluid of a meningitic case can be differentiated from that of a case of poliomyelitis by the larger protein content and by the predominance of polymorphonuclear cells. The etiological organism is usually present.

Epidemic Cerebrospinal Meningitis. The encephalitic type of poliomyelitis is likely to be mistaken for epidemic cerebrospinal meningitis, as the symptoms of vomiting, headache, rigidity of the neck, high temperature, and drowsiness usher in both diseases.

Examination of the spinal fluids, which will reveal the presence of the meningococci, as well as a study of the clinical symptoms, will clear up the diagnosis.

The main points of distinction in the clinical picture of the two diseases are: The continuance of a high temperature with an intermitting curve in epidemic cerebrospinal meningitis, in contrast to the abatement of the fever before paralysis occurs in cases of poliomyelitis; the spasticity in epidemic cerebrospinal meningitis, in contrast to the flaccid condition in infantile paralysis; the inability of the patient to move his head in any direction in meningeal cases, whereas in poliomyelitis, he can turn his head sidewise in spite of the rigidity present; and the petechial eruption, if present, in epidemic cerebrospinal meningitis.

Diseases Presenting Pseudoparalysis or Spasm.—*Scurvy.*—This condition may suggest poliomyelitis, if the extremities become immobilized from fear of pain. Examination shows that the child can move his limbs, and that the reflexes are normal. Other positive findings of scurvy are swelling along the long bones and bleeding of the gums.

Rachitic Paralysis.—The loss of power in this condition is due to general atrophy and is not of paralytic origin. The condition of the reflexes and the presence of symptoms characteristic of rickets aid in clearing up the diagnosis.

Spastic Paralysis or Tetanoid Pseudoparalysis.—It is not difficult to differentiate tetany from poliomyelitis. In the former, there is a symmetrical spasm of the muscles of the hands and feet, the reflexes are exaggerated, and the muscles can be made to contract by percussing the nerve.

Hysteria.—This disease is characterized by anesthesia, sensory disturbances, convulsions, and paralysis—symptoms suggestive of poliomyelitis. Hysteria rarely affects children, being a disease mainly of young women. The normal condition of the reflexes is indicative of hysteria. Hoover's sign is also of value in diagnosing feigned paralysis.

Conditions Presenting Loss of Power Due to Lesions Other Than Poliomyelitis.—*Congenital and Traumatic Conditions.*—When a physician is consulted regarding the loss of power in an extremity, he must first exclude congenital and traumatic conditions as the cause.

Congenital club-foot can be diagnosed by the absence of symptoms of paralysis. *Congenital dislocation of the hip* is characterized by the peculiar gait. The diagnosis can be confirmed by physical examination and by radiograms. *Spina bifida* may present a paralysis that closely simulates the paralytic phase of poliomyelitis. The presence of sensory disturbances will help to differentiate the diseases.

The traumatic etiology to be considered includes sprains, fractures, birth injuries, and pressure on nerves. Any injury involving a sprain or fracture should be carefully recorded in the history, since pain and loss of power may be traced directly to a trauma. On the other hand, the possibility of the occurrence of poliomyelitis as a secondary infection following an injury should not be overlooked.

Parturient paralysis should also be considered, because this shows a lower motor neuron involvement such as is found in poliomyelitis. The history of the case recording an injury at birth, and the duration of the involvement, point toward a birth trauma; on the other hand, the presence of paralysis elsewhere and an acute onset indicate poliomyelitis.

Pressure on a nerve sometimes causes it to lose power temporarily, but this condition is usually accompanied by a tingling in the limb.

Tuberculosis of the Hip.—The pain and limp present in a case of early tuberculosis of the hip may cause one to mistake the disease for infantile paralysis. The insidious approach of tuberculosis, the muscle spasm, and the fixation of the joint from pain and not from paralysis should prevent confusing tuberculosis with poliomyelitis.

Bell's Palsy.—This disease presents a facial paralysis, accompanied by earache, and usually follows exposure to cold. A spinal puncture settles any doubt as to the disease in question.

Pott's Disease.—The paralysis in this condition has led to confusion with poliomyelitis, but the nature and onset of the two diseases should prevent any errors.

Pseudohypertrophic Paralysis.—Such a paralysis is easily differentiated from poliomyelitis by the marked hypertrophy of the muscles.

Hereditary Ataxia.—The chronic course of this affection, the presence of several cases in a family, and the contracture deformities without paralysis aid in diagnosis.

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CHAPTER VIII.

PROGNOSIS IN GENERAL.

It is a difficult problem to establish the prognosis in poliomyelitis as the physician is requested not only to estimate the immediate danger, but also to forecast the possibility of the occurrence of paralysis. He will be asked also to predict both the extent of the paralysis and the probability of recovery. In early times, it was thought that the disease always resulted in paralysis, and that a fatal outcome was rare; today, we appreciate the fact that paralysis does not necessarily occur and that death not infrequently results. We also know that when paralysis does occur, the return of good function is possible; the recovery may be the result of spontaneous improvement, or it may be brought about by the use of the many conservative and operative measures at our disposal.

THE PROGNOSIS AS TO LIFE.

The prognosis as to life is based upon the location and extent of the pathology, and upon the occurrence of complications, particularly that of pneumonia. The outlook is also influenced by the age of the patient, the severity of the symptoms in the early stages, and the mental status of the child.

In most cases, death follows simultaneous involvement of the respiratory centers, and the patient usually dies within seventy-two hours of the onset of paralysis. While the outlook is not so grave, and recovery may take place when the intercostal centers alone are involved, still such cases are prone to develop bronchopneumonia, which as a rule proves fatal. Unfortunately, there is no way of telling if the respiratory centers will become involved, since no one can foresee where the paralysis will localize, or to what extent it will advance. In general, the higher the lesion the greater is the danger that the respiratory centers will become paralyzed. Peabody, Draper, and Dochez[1] found involvement of the cervical cord in all fatal cases. In ascending poliomyelitis, which resembles Landry's paralysis, the prognosis is grave while the paralysis is progressing, and when breathing becomes abdominal with the chest muscles fixed, death is certain.

The prognosis is also affected by the extent of the cord pathology. The greater the area of destruction, the graver is the outlook.

We have already spoken of the possibility of a fatal outcome from the development of pneumonia following paralysis of the respiratory muscles. Death may result from other complications, such as convulsions with no respiratory involvement and cardiac failure with medullary involvement. Diseases, such as enterocolitis, noma, and pertussis may also complicate the condition, making the outlook grave.

There are two conditions that tend to make the prognosis as to life more encouraging. In most cases the initial attack represents the maximum paralysis. If there is no further development of weakness within six to twelve hours, the paralysis rarely advances. Then, too, the lesion may be in close proximity to the respiratory centers, and yet not involve them.

The outlook as to life seems better in young children than in infants under one year of age, or in older children or adults. Wickman[2] found the fatality-rate among patients under eleven years of age to be 11.9 per cent, and among patients between the ages of twelve and thirty-two years, 27.6 per cent. While there have been exceptions to these rates, they have not been so numerous as to influence the general opinion that children in the first five years of life are more likely to recover.

Severe illness and excessive cerebration during the first few days are grave signs. Peabody, Draper, and Dochez[1] found that cases that proved fatal were usually very ill and prostrated at the beginning of the attack. Continued cerebral symptoms are of grave significance. There is usually no decline in temperature with the onset of paralysis in cases that result in death. A very rapid pulse, between 135 and 140, and respiration between 55 and 60 are discouraging symptoms.

Peabody, Draper, and Dochez also have found that the mental condition of the patient is a factor upon which to base the prognosis of the disease. The stuporous and irritable cases rarely proved fatal, but alert cerebration on the part of a child was of serious import. A careful distinction must be made between this state of alertness, which is a discouraging symptom, and irritability, which is a favorable sign.

Death is unlikely after the eighth day of muscle weakness. Wickman[2] found that death usually occurred on the fourth day of paralysis, with limits from the third to the seventh day after the loss of power was noticed. Peabody, Draper, and Dochez,[1] figuring from the onset of the disease, found the fifth day to be the most fatal, with limits from the fourth to the eighth day. Death after the eighth day usually is due to complications.

The prognosis as to life, when based on mortality statistics of past epidemics, is somewhat misleading, as such data included only cases that had been diagnosed from the paralytic symptoms. The abortive type, of which there were undoubtedly a great many cases in each epidemic, was not recognized. It is also true that many cases of slight paralysis may have been entirely overlooked. When such allowance for error is made, the prognosis as to life is more favorable than the mortality-rates, outlined below, indicate.

The fluctuation in the death-rate in different epidemics is particularly striking. Wickman[2] reported a fatality-rate of 16.7 per cent in one series of 868 cases, and a rate of 42.3 per cent in another of 26 cases. Investigators of other epidemics in both Europe and America have found that the rate varied from 10 to 25 per cent, or even higher. The average rate was about 15 per cent.

The mortality was also found to vary in different foci of the same epidemic, or even within a single focus. Some epidemics showed a tendency for the late cases to assume a higher degree of mortality, due possibly to the increase in virulence as the infection progressed. It was also noted that the rates in epidemics occurring at different times in the same locality fluctuated, as shown by the mortality of 5 per cent in the New York City epidemic of 1907 and the rate of 26.9 per cent in the 1916 epidemic in the same city.[3]

All these statistics are based upon the occurrence of poliomyelitis in epidemic form, as sufficient sporadic cases have not yet been investigated and studied to permit passing judgment upon their mortality.

THE PROGNOSIS AS TO PARALYSIS.

There is no way of anticipating the development of paralysis. In fact, a great many cases of poliomyelitis do not suffer paralytic lesions. The clinical symptoms of pain, loss of tendon-reflex, and muscular twitching in an extremity are of little prognostic value, as they appear in cases in which no paralysis subsequently occurs. The reflexes may be absent, recurrent, or exaggerated, and yet paralysis does not develop. There is no relation between the severity of the systemic symptoms and the development of paralysis. Frequently the cases of mildest onset will have an extensive loss of power, while those with severe prodromal symptoms will suffer slight paralytic lesions.

Draper[4] believes that the cell-count of the spinal fluid is a help in prognostication. In a large number of observations he found that paralysis rarely developed if the cell-count was under 100 per cmm. during the first twelve to eighteen hours after meningeal invasion.

The subsequent condition was at the most only a transient facial paresis. Patients with a cell-count of 200 to 500 per cmm. usually became paralyzed, and cases having a count of 700 to 1000 cells per cmm. during the early hours proved fatal. Exceptions to these general observations occurred, but in very few cases that developed subsequent paralysis was the spinal fluid count below 100 cells per cmm. during the first twelve to eighteen hours.

Paralysis may occur on any day of the disease, but it usually appears on the third or fourth day, and seldom occurs after the eight day. There have been exceptional cases in which paralysis appeared after a long period, but these are not sufficiently numerous to make one apprehensive of paralysis after the eighth day. As a rule, the initial lesion is final, but cases of reinfection are not unknown. The writer has observed several cases of reinfection which were followed by attacks of paralysis.

THE PROGNOSIS AS TO RECOVERY OF FUNCTION.

After the onset of paralysis, the parents experience the greatest apprehension for the future condition of their child. Unfortunately, there are no means by which the physician may judge the ultimate outcome as to function. Recovery is directly dependent upon the cord pathology and upon proper treatment. If all the nerve cells governing a muscle have been destroyed, it is impossible for that muscle to recover function; if the nerve cells are only temporarily paralyzed from pressure, the muscle will recover almost complete power when the edema has been absorbed; if only part of the nerve cells have been totally damaged, a proportionate recovery may be expected in the muscle. Since some relief from pressure always takes place, the physician may safely predict an improvement in the condition. It is impossible to say how long this period of spontaneous improvement lasts, or what is its extent. The recovery may be rapid during the first few days or weeks, due to the absorption of the inflammatory edema. In such cases the outlook as to ultimate recovery is favorable. In other instances the paralysis may disappear gradually. When there is loss of power in the extremities, several weeks may pass before improvement is noticeable. In mild types the paralysis may last only a few days, seven to ten days being the usual duration; in others, recovery may be complete in six weeks. Improvement may be rapid for several weeks and then cease entirely. After six months, there is little return of muscle power, and the future disability may be fairly well estimated. Generally, the more extensive the cell damage, the slower is the process of recovery, and the greater is the danger of permanent paralysis.

The prognosis depends to a great extent upon treatment. The parent should be made to realize the importance of proper care. Definite improvement may be expected in the large majority of cases treated properly. Even when a large part of the body is paralyzed, the patient may recover some function under careful treatment. The factors favoring recovery, entirely independent of the cell-repair, are:

1. Initial rest.
2. The prevention of deformity and muscle-stretching.
3. Careful massage and muscle training, instituted at the proper period.
4. Mechanical treatment and operative interference, as indicated in the individual case.

The best results are obtained when treatment is begun early and carried out thoroughly for two years or more. In general, the prognosis is better under treatment in the first year, and the greater the voluntary return of power, the better the outlook. Early neglected cases should not be disregarded, for they are capable of much improvement, even after years of disability. In the writer's experience, a case of extreme equinovarus of over twenty years' standing was restored to perfect function by means of manipulation followed by tenotomy of the tendo Achillis and the plantar fascia. Upon the restoration of the foot to normal position, the anterior group of muscles recovered full power.

In cases in which deformity has occurred through lack of proper treatment, conservative and operative procedures may be used to secure correction. In general, no operative interference should be instituted until two years after the attack; exceptional cases are those in which contracture and deformity cannot be corrected by conservative and mechanical means.

As regards complete functional recovery, it is impossible to make a definite statement. If one considers the large number of abortive cases that totally recover, then the percentage of complete recoveries is very high. Undoubtedly, the number of total recoveries is greater than is generally estimated. Evidence of such recoveries is found in the large number of cases that apparently have completely recovered, but that years later show the effects of the attack in a deformity such as a mild claw-foot. In the epidemics in which the cases of complete restoration of function have been noted, the number has been found to vary extensively. Wickman[2] analyzed 530 cases from one to one and a half years after the acute attack, and pronounced 44 per cent cured and 56 per cent paralyzed. Of 212 cases in the epidemic of 1907 in Massachusetts, investigated by Wood,[5] 27 per cent were considered wholly recovered. In the Massachusetts epidemic of 1910, 16.7 per cent complete recoveries were recorded. In other epidemics, the percentage has been as low as 5.3 per cent. In the New York epidemic of 1916, 1233 out of 3441 cases, treated less than two months, were discharged with no visible paralysis, making 32.6 per cent total recoveries.[3]

As a rule, if complete return of function is to be the result, power will have returned in six months. Complete recovery may take place in the latter part of the first year, or even later, in cases in which deformity has been corrected, and the muscles properly treated. The usual result is not complete recovery of the muscle, but partial recovery with nearly complete restoration of function, a condition that simulates absolute restoration very closely.

THE PROGNOSIS AS TO SHORTENING.

Not infrequently the surgeon will be asked to give a prognosis as to shortening. As a rule, it is not extensive, the ultimate shortening being usually under 2 inches. While the shortening is often in proportion to the severity of the paralysis, still there are many cases in which this is not true. The writer observed long ago that many severe and extensive paralytic cases developed only 1 to 2 inches of shortening, whereas cases of paralysis of a single group of muscles had as much as 2 to 4 inches. Analysis showed that the patients who used the leg well in walking developed less shortening than those who used it poorly. It was apparent that insecurity or instability of the foot caused the patients to hop quickly over the weak leg, using it as little as possible. This explains why calcaneovalgus is associated with more shortening than one would expect to find in a single group paralysis; and conversely, why almost flail legs, actively used, show less shortening. Braces and stabilizing operations are often necessary to correct deformity, in order that by bearing weight and using the limb actively, the shortening will be minimized.

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PART II.

TREATMENT.

CHAPTER IX.

THE GENERAL TREATMENT OF POLIOMYELITIS.

THE treatment of poliomyelitis probably has incited more interest in the various branches of the medical field than the treatment of any other disease. The clinician, the pediatrist, the neurologist, and the orthopedic surgeon, all have sought to increase our knowledge of the lesion. In addition, experimenters have tried diligently to discover a means by which to prevent the development of paralysis, or to arrest its progression. From the intensive study that has been made, the treatment of poliomyelitis has evolved into four forms, the symptomatic, the preventive, the mechano-therapeutic, and the operative. Symptomatic treatment is carried out throughout all stages of the disease, but it is applicable particularly to the acute febrile period, at which time the disease is manifested by the symptoms of fever and pain and by meningitic disturbance. Preventive treatment is used during the acute stage in the hope of checking the onset of paralysis, and during the early paralytic stage in the hope that the paralysis will disappear, or cease to progress. Mechano-therapeutic treatment is carried out mainly during the early paralytic period, at which time the muscles are recovering. The operative treatment is used chiefly in the late paralytic period, when deformities have become more or less fixed, and when muscle unbalance and joint instability persist.

TREATMENT IN THE ACUTE FEBRILE STAGE.

Symptomatic Treatment. When a child presents symptoms suggestive of poliomyelitis, he should be put to bed in an isolated room. If the case is severe, the patient is so prostrated that he is obliged to remain in bed, but if the case is mild it may be necessary to insist upon his resting. Absolute quiet is essential, for noise and excitement tend to irritate the patient.

The diet should be regulated carefully as in any acute febrile disturbance. Light foods and forced liquids are advisable. It is well to give a cathartic, such as calomel, to free the intestinal tract of any possible infectious agent. The patient also should have plenty of fresh air and sunshine.

Preventive Treatment; Serum Therapy.—Our knowledge of the clinical aspect of infantile paralysis makes it possible to diagnose many cases of epidemic poliomyelitis before paralysis occurs, and to identify even sporadic cases in the preparalytic stage with a fair amount of accuracy. The recognition of the disease in the early stage affords the opportunity to attempt to prevent the development of paralysis.

Various investigators, basing their opinions upon the discovery that one attack of the disease renders the monkey immune to reinoculation, have suggested some form of serum as the preventive means.

The Experimental Basis of Serum Therapy.—The use of serum in poliomyelitis followed the discovery by Flexner and Lewis[1] that one attack of the disease in monkeys afforded immunity to reinfection. This resistant power was later demonstrated by Römer and Joseph[2] to be due to the presence of antibodies in the blood of the monkey, which in neutralization tests were found to exert a destructive action upon the virus. Flexner and Lewis,[3] and Levaditi and Netter[4] discovered that human serum, derived from individuals who had passed through an attack of the disease, also possessed neutralizing power for the virus. Experiments were then made to determine the effect of the serum derived from recovered monkeys or humans upon the development of the disease. Flexner and Lewis[5] showed that subarachnoid injection of the serum into monkeys, begun twenty-four hours after intracerebral inoculation of the virus, and repeated for several days, may either prevent the development of the disease, or delay the onset of paralysis.

One must bear in mind in considering this experimental work, that poliomyelitis in monkeys presents several characteristics that differ from the manifestations of the disease in man, differences indicating that the injection of serum should be more efficacious in humans. Experimental poliomyelitis is more severe and more frequently fatal than human poliomyelitis. Experimental poliomyelitis develops with great rapidity, paralysis appearing within twenty-four or forty-eight hours; in human poliomyelitis the prodromal period is of longer duration, lasting generally from two to four days.

The Use of Serum in Human Cases.—The first application of serum in the treatment of human cases was made by Netter.[6] He reported the use of human convalescent serum in 32 cases. When possible, the serum was obtained from persons who had had the disease within five years. It was withdrawn aseptically, controlled by a Wassermann reaction, and sterilized by tyndallization. Five to 13 cc. of serum were injected into the spinal cavity daily for eight to fifteen days. In 6 cases recovery was complete and rapid; 15 cases showed varying degrees of improvement, but the value of the serum in producing the change was questionable; in 3 cases the progress of the disease was not affected; and 8 patients died. Netter concluded from his results that the intraspinal injection of human convalescent serum is decidedly beneficial. He believes that injections of serum in the preparalytic stage may prevent the onset of paralysis, and that injections in the early paralytic stage may cause the paralysis to disappear, or to remain stationary.

The epidemic of 1916 afforded opportunity to use serum treatment in a number of cases. Amoss and Chesney[7] reported 26 cases of intraspinal and intravenous, or subcutaneous, injection of human serum, obtained from recovered and convalescent cases. In 9 of 12 cases that were paralyzed before administration of the serum, there was no further progression. Of 14 cases in which the serum was injected before any paralysis was apparent, 10 recovered without a trace of muscular weakness; 2 died from respiratory failure; and 2 cases developed some paralysis. The results were so promising that Amoss and Chesney advised the administration of at least 50 cc. of serum in every case within thirty hours of the onset if possible. In their opinion, the serum should be injected intraspinally and directly into the veins or into the subcutaneous tissue.

Schwarz[8] treated one series of 21 cases with convalescent blood serum, and another series of the same number by expectant methods. Nine of the first series, and 17 of the second, developed paralysis. Schwarz concluded that not too much was to be expected from serum treatment, although he admitted that his series was perhaps too small to permit him to draw definite conclusions.

Zingher[9] treated 54 cases in the preparalytic stage, and 119 in the paralytic stage, with immune human serum. Injections were made intraspinally in doses of 15 cc., and repeated every twenty to twenty-four hours until two or three administrations had been made. Of the 54 cases treated in the preparalytic stage, 44, or 82 per cent, did not develop paralysis, and of the 10 cases in which paralysis occurred, 5 made a complete recovery. Of the 119 cases treated in the paralytic stage, 45 died. This high mortality is undoubtedly explained by the serious condition of many of the cases at the time of treatment. While Zingher considered that his group of cases was too small to permit his drawing definite conclusions as to the value of the serum, he believes the treatment to be beneficial and recommends it.

Peabody[10] treated 51 cases in the preparalytic stage, and 60 cases in the early paralytic stage, by intraspinal injection of convalescent serum taken from patients who had had the disease from three to forty years previously. The number of injections in the preparalytic cases depended upon the clinical picture, 27 of the patients receiving one treatment, 16 patients two, 6 patients three, and 2 cases four treatments. The majority of cases were treated within forty-eight hours of the onset of the disease. Thirty-five, or 69 per cent, of these cases recovered without paralysis; 5 cases, or 10 per cent, died; and 11 cases, or 21 per cent, became paralyzed, the condition being slight in 6 of the cases. It is difficult to judge just what effect the serum had on the course of the paralyzed cases, as in some instances the cessation of the extension of the paralysis may have been spontaneous. Roughly, 12 per cent showed definite rapid improvement; 51 per cent did not improve, or grew worse; 32 per cent died; and in 3 cases, or 5 per cent, the results are unknown. Peabody recommends the use of serum in the preparalytic stage, but does not advise its administration in the paralytic stage unless the disease is progressing, for it might increase the pressure in the spinal cord.

Draper[11] treated 45 cases in the preparalytic stage with human convalescent serum injected intraspinally. The results were such that Draper could draw no definite conclusions as to the value of the serum. Paralysis developed in some cases, but in others there was no sign of weakness. There were more instances of partial paralysis among the cases in which the serum was administered, than among the untreated cases. The serum did not seem to play any part in delaying the development of the paralysis. Draper concluded that although statistically the value of serum therapy is not convincing, yet clinically the treated cases show that the serum is beneficial.

Taylor[12] treated two series of cases, one of 19 patients and the other of 8 patients, by intraspinal, subcutaneous, and intravenous injections of serum. Eight of the first series recovered completely; in 6 cases there was no further progress of paralysis and some improvement; in 3 cases which were markedly paralyzed when treated, the reaction was negative; and 2 patients died. All 8 cases of the second series recovered completely. Taylor concluded that injection of serum after the acute febrile stage, when paralysis has definitely developed, is without value.

Wells[13] used immune human serum in 15 cases, injecting it intraspinally, intravenously, intramuscularly, singly or in combination. The dose of the intraspinal injection ranged from 5 to 10 cc., that of the intravenous from 8 to 50 cc., and that of the intramuscular from 6 to 30 cc. After intravenous and intramuscular injections, from 5 to 15 cc. of spinal fluid were withdrawn to reduce pressure and favor circulation in the central nervous system. In all cases, a noticeable improvement occurred following intravenous and intraspinal injection, but unfortunately it was only transitory in some instances.

Place,[14] in reporting on statistics of 200 cases of poliomyelitis treated by various kinds of sera and drugs, made mention of 40 cases that were injected with immune serum in the paralytic stage. Fifty-two per cent of the 40 died; the paralysis progressed in 33 per cent; and extension of the paralysis ceased in only 15 per cent. Too much weight must not be placed upon these results, since only the most hopeless cases were treated. Place also reported 8 cases that were treated with immune serum in the preparalytic stage; 3 of this number developed paralysis. This series was compared with a series of 16 cases that were not treated with serum, and in which only 1 patient developed paralysis.

Petty[15] administered serum in 11 cases by the subdural route. He drew the following conclusions: Early administration of the serum is preferable; injection should be made by the subdural route by gravity; and the amount of serum introduced should be less than the quantity of spinal fluid withdrawn.

Sophian[16] reported a small series of 4 cases in which serum, obtained from 2 adults who were three weeks convalescent, was used. One case treated in the preparalytic stage recovered without paralysis, but the case may have been of the abortive type. Of the 3 other patients who were treated in the paralytic stage, 2 showed no improvement, and although the remaining case improved, Sophian did not believe the improvement was the result of the serum.

Preparation of the Serum.—The preparation and administration of the serum, as outlined below, is based chiefly upon the treatment carried out in the Health Department Hospitals of New York City during the epidemic of 1916,[17] and upon the work of Amoss and Chesney[7] who have experimented widely in the use of serum.

It is generally agreed that the serum from cases of poliomyelitis of recent date, that is, from two to six months convalescent, is most potent. As a rule, the neutralizing power is found to weaken with age, although serum from cases of even thirty years' standing has been found to contain immune bodies.

The blood of the donor of the serum and that of the patient must be complementary. A Wassermann test should be given to the individual from whom the serum is to be obtained. The amount of blood withdrawn varies with the weight, general health, and age of the person. Five hundred to 700 cc. is the average amount taken from adults, and 200 to 250 cc. from children of nine or ten years of age. After two or three weeks' interval, similar amounts may again be obtained. The blood is withdrawn by using a No. 15-gauge platinum or steel needle. In children, or in stout persons with small veins not easily seen, it may be more satisfactory to use a No. 17-gauge needle attached to a 1-ounce Record syringe. The blood is collected in small glass bottles in quantities of 1 or 2 ounces, and given a long slant, so as to obtain as long a surface for the serum as possible. The blood is allowed to cool, and then the bottles are placed in the ice-box to allow the serum to separate from the clotted blood. After twenty-four hours, the serum is inactivated at 55° C. for one hour, passed through a Berkefeld stone filter, and transferred to small sterile bottles. The serum should be free of hemoglobin, red blood cells, or any solid matter. It should be kept in the ice-box until needed. Some users of serum recommend the addition of 0.2 per cent of tricresol as a preservative, but others believe that this compound increases the local irritant action of the serum. It is the writer's opinion that the serum will be effective provided it is fresh, obtained under sterile conditions, and kept in the ice-box until required.

The Administration of the Serum.—The serum should be administered in the preparalytic stage, even when only systemic symptoms are present. The writer does not believe it well to wait for any signs indicative of involvement of the central nervous system, for very often it is impossible to tell when the meninges have been affected. It is, of course, easier to determine upon the administration of the serum, if there is a case of infantile paralysis in the family or in the neighborhood. In all cases, however, whether epidemic or sporadic in form, an intraspinal injection of serum should be made when a child presents a clinical picture of poliomyelitis and a lumbar puncture confirms this diagnosis. If the lumbar puncture is made so early in the disease that the spinal fluid is negative, a large dose of serum may be administered by the intravenous or subcutaneous route. Another puncture should be made in ten to twelve hours. If the fluid then shows pathological changes, an intraspinal injection of serum and another intravenous injection should be made.

Before injection the serum should be warmed to the body temperature. In intraspinal injections, the amount of serum administered varies according to the quantity of spinal fluid withdrawn and according to the pressure. The amount of serum should never exceed the quantity of fluid withdrawn. In intravenous injections, 40 cc. of serum are administered in children up to two years of age, and larger amounts in older patients.

The best method of injection seems to be the combined intravenous and intraspinal. Since the serum is weak in antibody content, it is important that as large a quantity as possible should reach the pathological center. By the use of both routes, it is possible that more serum will penetrate into the body and reach the site of the lesion.

There is some question as to whether serum, injected by the combined intraspinal and intravenous method, ever comes in contact with the virus. In view of the presence of immune bodies in the human convalescent serum, it would seem that the results of the injection of this fluid should be more satisfactory. The localization of the lesion in the central nervous system may account for the failure of the serum to be more effective. It is a well-known fact that it is difficult to treat effectively, by chemicals or sera, any disease in which infection is located in the nervous system. It is also true that once the virus has become localized in the central nervous system, it is not found again in the blood or in the cerebrospinal fluid. Therefore there is a possibility that the serum, injected into either the spinal fluid or into the blood, may not come in contact with the virus.

Amoss and Aycock[18] are still working on the problem of the introduction of serum. Their study is based upon the circulation of the fluids of the central nervous system, and upon the experimental production of changes in the currents of these fluids. Aycock and Amoss explain the inability of the serum to reach the inflammatory center in the gray matter of the cord as follows: The flow of fluid within the nervous tissue itself is probably from the capillaries along the pericapillary, perineural, and perivascular spaces toward the subarachnoid space. If the serum is injected into the subarachnoid space, it will not penetrate to the site of the lesion in the anterior horns. Also, the walls of the capillaries of the nervous system have a discriminating power and prevent the passage of the serum from circulation to the perivascular system. The control of the capillaries is greatest in the vessels of the brain and spinal cord, and in the choroid plexus. Flexner and Amoss,[19] and Mott[20] have studied in detail the power that the choroid plexus exercises over the composition of the cerebrospinal fluid.

Attempts have been made to break down the barriers preventing the penetration of the serum to the pathological lesion. Flexner and Amoss have shown that when the integrity of the meninges or of the choroid plexus is disturbed, the control of the choroid plexus over the composition of the cerebrospinal fluid is diminished. In one experiment, in which a state of inflammation was set up in the meninges, the antibodies that had been injected into the blood passed into the cerebrospinal fluid. In another experiment, in which horse serum was injected into the subarachnoid space, the virus was found to pass into the central nervous system. Experiments have been made with hypertonic solutions in the hope of producing changes favorable to the circulation of the serum.

The Use of Hypertonic Solutions.—Weed and his co-workers[21] have shown that intravenous injection of hypertonic solutions in normal animals causes a reduction in the volume of the brain and spinal cord, with a marked lowering of the cerebrospinal fluid pressure, and an aspiration of fluid from the subarachnoid space into the perivascular lymph spaces of the brain and cord. Aycock and Amoss[18] obtained similar results in experimenting with edematous cords of monkeys, sacrificed in the acute stage of poliomyelitis. These monkeys, which were given convalescent human serum intraspinally and intravenously, and hypertonic salt solutions intravenously, showed marked improvement as compared to the controls. Whether the injection of hypertonic solutions is beneficial in human cases may only be conjectured. So far, the method has been tried in only a few human cases, and a report has been published of only one case. In this case, one of the type resembling Landry's paralysis, Aycock and Amoss injected hypertonic solution immediately after the administration of human convalescent serum. The respiratory organs were already involved when the hypertonic solution was administered. No intravenous injection was made. Aycock and Amoss hesitate to judge just what effect the injection of hypertonic solution had upon the edema and upon the flow of the human serum, but apparently the procedure was harmless. The patient recovered and was continuing to improve when last seen.

The injection of a hypertonic solution produces certain changes, which are advantageous to the circulation of the serum. (1) The intravenous injection of a hypertonic salt solution, by shrinking the central nervous tissues, makes more space available in the subarachnoid area for the entrance of large amounts of serum. (2) The favorable change produced in the tonicity of the blood, with marked decrease in the volume of the brain and spinal cord, reduces the inflammatory edema of the cord. Since edema may not be explained entirely on the basis of osmosis, further experiments are needed to make the condition clear. Possibly the most important benefit derived from the injection of hypertonic solution is the aspiration of serum from the subarachnoid space into the perivascular system, thereby bringing the serum nearer the central nervous system. Another possible advantage is the fall in the cerebrospinal fluid pressure. This loss is replaced by fluid from the blood stream, and if immune serum has been injected intravenously, it will pass with the fluid from the blood into the fluid spaces of the central nervous tissue.

The Use of Immune Horse Serum.—Rosenow[22] and his co-workers claim to have developed a curative serum by injecting strains of the pleomorphic streptococcus into the horse.* Rosenow has experimented widely with this serum, and reports good results. One hundred and sixteen cases were treated in the epidemics at Davenport and Dubuque, Iowa, in 1917 and 1918; and 128 cases were treated in various parts of the United States, during the summers of 1918 and 1919, by physicians to whom the serum was sent. Sixty of these cases, in which the serum was administered in the preparalytic stage, recovered without residual paralysis; of 61 cases that had slight paralysis at the time of treatment, all but 1 recovered completely; of 123 patients with advanced paralysis when treated, 61 recovered completely, 18 died (11 having had symptoms of involvement of the medulla when treated), 30 had residual paralysis, and 14 results were not known. The prognosis as to death and as to the recovery of function, in both the epidemic and sporadic outbreaks, was far better in the treated cases than in those not treated.

Rowan[23] used horse serum in a series of 85 cases. Ninety-five per cent of the series recovered completely.

Serum, prepared in the horse, was also used by Nuzum,[24] but his method of preparation of the serum differed from that of Rosenow. Nuzum used for his immunization process strains of streptococci, which he claimed to have cultivated from the cerebrospinal fluid.

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* See Chapter on Etiology.

Nuzum and Willy,[25] experimenting with this serum, have found it effective in neutralizing the virus *in vitro*, and *in vivo* when injected intraspinally or by the combined intraspinal, intravenous, and intramuscular routes.

While the results published by these experimenters are impressive, a larger series of observations, carefully controlled, must be made, before definite conclusions can be drawn as to the value of horse serum. There are many details that are questionable in the preparation and use of both Rosenow's and Nuzum's sera. The etiological organism of streptococcic origin has not been generally accepted.* Furthermore, Nuzum's success in cultivating a streptococcus is most exceptional; no other bacteriologist has been able to determine the presence of bacteria in the cerebrospinal fluid. Attempts to produce an antiserum by immunizing the horse with the virus have not been successful. There is also some objection to using serum from animals, since they are so susceptible to other diseases. Amoss and Eberson[26] found that intraspinal injection of Rosenow's serum, or of that advocated by Nuzum and Willy, failed to protect the monkey against experimental infection produced by injecting the virus intravenously. On the other hand, serum from recovered monkeys, when introduced under the same conditions as the horse serum, afforded perfect protection.

Other investigators who seem to have been successful in producing an antipoliomyelitic horse serum are Neustaedter and Banzhaf.[27] Their experiments were based upon the possible reaction of the horse to an endotoxin, which they prepared by digesting the virus in the filtrate of a brain and cord emulsion with a proteolytic ferment. Five injections of the endotoxin were made into a horse; two weeks later serum was prepared from the blood. Neustaedter and Banzhaf have made five neutralization experiments with the horse serum in monkeys, and all were positive. The serum was injected into three human cases with success. The series treated is too small to recommend the general use of the serum, but Neustaedter and Banzhaf believe their successful results warrant its use, particularly if human serum is not available.

The Use of Normal Sera.—Normal serum, whether of human or animal source, is supposed to be of value in producing a polynuclear leukocytosis in the spinal fluid. Normal human serum has been used by Rueck[28] in 3 cases, by Roby[29] in 2 cases, by LeBoutillier[30] in a few instances, and by Zingher[9] in 43 cases. Rueck treated his patients by transfusion of blood taken from healthy adults and prepared with 2 per cent sodium citrate solution. His results were so favorable that he has firm belief in the curative action of the normal adult blood. Roby treated 2 cases by intramuscular injection of citrated whole blood. He was unable to drawn conclusions as to the value of the serum. LeBoutillier also reached no conclusion from his experience. Zingher used the normal human serum in 10 cases in the preparalytic stage, and in 33 cases in the paralytic stage. Of the 10 cases, 9 recovered without paralysis and 1 died; of the 33 cases, 4 recovered completely, 24 recovered with partial paralysis, and 5 died. Zingher's results were such as to lead him to advise the use of normal serum in preference to immune serum in the preparalytic stage.

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* See Chapter on Etiology.

Sophian[16] and Place[14] experimented with normal horse serum. Sophian used the serum in 10 cases; it was of some benefit to those in which it was used early. From a comparison of his results with those obtained in 4 cases treated with immune serum, he concluded that horse serum was as efficient as human convalescent serum. Place did not obtain favorable results.

The series of cases treated with normal human and animal sera have been so small, that it is impossible to draw definite conclusions as to their value.

Autotherapeutic Treatment.—Duncan[31] reported cases in which 1 cc., or less, of cerebrospinal fluid was taken from the patient himself, and injected into the tissues. The cases improved. The value of this treatment is questionable since the virus is not found in the spinal fluid. Duncan's faith in the method is founded upon the fact that autotherapy, which has been found effective in toxic neuritides, should be of value in poliomyelitis, a disease of the same nature.

Conclusions.—At the present time it is impossible to judge the efficacy of any kind of serum, whether normal or immune in form, or of animal or human source. The series of cases in which sera have been used have not been sufficiently large to permit us to draw any definite conclusions. Also, no accurate comparisons of the results of a series of treated and untreated cases have been made. Whether the serum is effective in preventing paralysis cannot be determined, since we do not know how many untreated cases escape paralysis. The early symptoms in no way prognosticate the course of the disease, and cases with severe initial symptoms or high fluid cell-counts may develop little paralysis. It is also impossible to judge whether serum has any effect upon the recovery from paralysis, since we do not know the degree of spontaneous recovery.

The results of serum treatment thus far have varied greatly.

Some experimenters, who have found it of benefit in a considerable number of cases, have become enthusiastic advocates of the treatment. The possibility that a large series of good results may contain many cases of the non-paralytic type must not be overlooked. One great advantage of the use of serum is that it has stimulated the diagnosis of the disease in its early stages.

Of all the kinds of sera that have been used, human poliomyelitic convalescent serum seems to be the most effective. Its use is based upon the accepted principles of immunity. If properly administered, it causes no disturbances. It is the opinion of the writer that a person, showing symptoms characteristic of poliomyelitis, should receive an injection of immune convalescent serum, obtained from an individual who had the disease from two to six months previously. To be effective, the serum must be administered in the early stages, since it has been found to be of no benefit once paralysis has occurred.

The viricidal action of this serum has not been definitely determined. Its power has been demonstrated only when the serum comes in contact with the virus. It is difficult to establish this contact, since the lesion is localized in the central nervous system to which serum, injected either into the fluid or into the blood, does not penetrate. The experimental work with hypertonic solutions, which Aycock and Amoss are doing in the attempt to overcome the mechanical obstacles that prevent the serum from reaching the virus is very interesting. Although no definite results have been obtained, the favorable effect that the treatment had upon the course of the disease warrants further experimental work along this line.

The Use of Drugs.—Drugs are used both as sedatives for restlessness and pain, and as a means of preventing the infection of the spinal cord by the pathogenic organism. Morphine, codein, aspirin, and similar remedies may be used to overcome the pain and restlessness. Among the drugs that have been used to combat infection are: Adrenalin, urotropin, quinine, and salvarsan.

Adrenalin.—Meltzer[32] is the main advocate of the use of adrenalin or epinephrine. He believes that intraspinal injections of this drug keep down the edema and inflammatory processes, thereby having a beneficial effect upon the nerve tissues. The quantity of adrenalin injected depends upon the severity of the case. Before the first injection, a quantity of spinal fluid is withdrawn, the amount depending upon the pressure prevailing in the spinal cord. When possible, injections are given at least every four hours, and continued for four to five days after paralysis has disappeared, or until the condition has remained stationary for some time.

As to the results obtained by users of this drug, some have reported beneficial effects, others have reached no conclusions as to its value, and still others have claimed it valueless. Among those who recommend the injection of adrenalin is Lewis,[33] who reported its use in 77 cases. Six to ten weeks after the onset of the disease, 21 cases had made complete recovery; 21 cases showed great improvement, and further progress was anticipated; and 17 of the cases were expected to result in permanent disability. Eighteen patients died, but most of them had been moribund when treated, and only 5 could be said to have died from poliomyelitis under adrenalin treatment.

Hoyne and Cepelka,[34] following the method of Meltzer, used adrenalin in early cases. Improvement occurred in some cases in one-half to one hour after intraspinal injection, although it was seldom permanent. In some cases gradual improvement seemed to be more rapid.

LeBoutillier[30] found that the injection of adrenalin chloride, following lumbar puncture, was of some benefit. Doses of 1 to 2 cc. were administered, depending upon the age of the child. Injection was made every twelve to twenty-four hours, since the doses given at shorter intervals caused marked disturbance. From his results, LeBoutillier questioned the effect of the drug in the paralytic stage, for if the lesion were high, he found it impossible for the solution to reach the upper part of the cord, even if the buttocks were elevated. In 2 cases of very severe chest involvement, in which a combination of adrenalin chloride and immune serum was used, marked improvement resulted. The adrenalin chloride was administered in the morning, and the immune serum at night. Until a better treatment is suggested, LeBoutillier recommends the use of adrenalin.

Weisenberg and Leopold[35] studied the reaction of adrenalin in a considerable number of the 717 patients admitted to the Municipal Hospital in Philadelphia during the epidemic of 1916. The injection had no effect upon the course of paralysis. A number of cases reacted badly, especially after the second or third injection. Weisenberg and Leopold were undetermined as to the value of the solution.

Among those who have found the treatment of no value are the members of the 1916 New York Health Department[17] who studied its use during the epidemic of that year, Evans,[36] and Place.[14]

Urotropin.—The experiment of Cushing and Crowe,[37] in which urotropin was found excreted into the subdural space, led Flexner and Clark[38] to test its value in animals suffering from poliomyelitis.

They found that the drug prolonged the incubation period, and possibly entirely prevented the onset of paralysis in a number of animals. Flexner then advocated the use of urotropin in human cases, the doses varying from 1 to 5 grains depending upon the age, and given every four hours during the day.

Quinine.—The New York Board of Health[17] reported 6 cases which were treated with quinine and urea hydrochloride injected intraspinally on the first, second, third, fourth, or fifth day of illness. It was concluded that the treatment had little effect upon the course and prevention of the disease, although it was not possible to judge its value in the preparalytic stage, as the 6 cases were paralyzed when treatment was begun.

Conclusions.—On the whole, drugs seem to be of little value. Urotropin, which tends to split up into formaldehyde, may help in counteracting the infection. The writer would administer urotropin in conjunction with sodium benzoid as a routine procedure.

TREATMENT IN THE EARLY PARALYTIC STAGE.

The early paralytic stage begins with the subsidence of the acute febrile symptoms, and lasts up to the time when the paralysis may be considered stationary. There are differences of opinion as to the duration of this period, but it is usually reckoned at about two years. The designation of this phase as two years is undoubtedly influenced by the fact that an effort is being made during this time to restore muscle power by the use of conservative measures. It is only when conservative means are not efficient in preventing the development of a deformity during the first two years, that recourse is had to operative measures.

The treatment in the early paralytic stage has three important phases: The first includes general symptomatic treatment; the second and most important is the prevention of the development of deformity; and the third phase is the restoration of muscle power.

Symptomatic Treatment.—The patient should be kept at rest during the early part of the paralytic period. When we consider the pathological condition throughout the central nervous system at this time, the importance of complete functional rest is evident. Inactivity aids Nature in her attempt to clear up the inflammatory condition and to restore the exhausted nerve centers. No massage or manipulation should be done during the early part of this period. Cases have been observed by the writer in which massage and exercise at this time were responsible for the persistence of paralysis. As soon as the treatment was discontinued, and the patient put to bed, there was a gradual return of power.

Pain and tenderness may be relieved by the application of heat, which promotes the general circulation by producing a hyperemia. The heat may be applied either in dry form, by baking, or in moist form, by hot fomentations or hot baths. In cerebral cases, ice-bags on the head and back of the neck will relieve the pain. If anodynes are necessary, aspirin, paregoric, or morphine may be given.

In respiratory involvement, every effort should be made to permit the breathing to be free and easy. Clothing and any constricting bands should be removed. Mild sedatives are indicated to relieve any restlessness, for excitability tends to make breathing more difficult. The active administration of oxygen is of very little value.

In cerebral and lumbar involvement, lumbar puncture is of therapeutic value in relieving the intracranial pressure. The puncture is often followed by distinct improvement. If necessary, the process may be repeated several times.

The use of serum treatment in early paralytic cases has already been described in connection with the application of serum in the acute period.

Deformity.—Deformity is the greatest factor with which we have to cope in poliomyelitis. We are dealing with a child who has his whole life before him, and whose mental outlook as well as physical welfare may be influenced by a defective physique. We should be eager to take advantage of any opportunity to prevent the child from becoming a cripple. Fortunately, in the treatment of poliomyelitis, there are measures at our dispoasl by which the *majority of deformities can be avoided.* The development of most deformities is due to neglect of these precautionary measures.

In order to avoid deformity, one must appreciate the etiological factors that enter into its formation, one must understand the pathological changes that take place in its development, and one must be versed in the forms of treatment by which deformities can be avoided.

The Etiology of Deformity.—The etiological factors in the development of deformity are:

1. Muscle unbalance.
2. Ligamentous and tissue contraction.
3. Habit posture.
4. Static influence.
5. Gravity.

Muscle unbalance is the chief etiological factor in the development of deformities. When paralysis occurs in one muscle group, as for instance in the anterior group in the leg, the muscles and tendons relax, while the opposing group, in the case of the leg the posterior or gastrocnemius, tends to contract. This contraction is the result of the shrinking of the fibrous and capsular tissue. As soon as the arc of motion of a joint becomes limited, the *ligaments* and *fascia* contract.

Owing to our knowledge of the effect of muscle unbalance, we are able to anticipate the occurrence of deformity, and when the actual group paralysis is recognized, we can predict the exact deformities that will develop. Listed below are the deformities that may be expected to occur as the result of the paralysis of the various muscles.[39]

Foot.

Varus	Peronei
Valgus	Anterior tibial Posterior tibial Flexor longus hallucis Both tibials Whole leg
Equinus	Anterior muscles paralyzed or weak Complete paralysis
Equinovarus	Anterior muscles with persistence of flexor longus hallucis Anterior and external group
Equinovalgus	Anterior and internal muscles Anterior muscles and weight-bearing
Calcaneus	Posterior muscles
Calcaneovalgus	Posterior muscles and one or both tibials

Knee.

Genurecurvatum	Posterior thigh muscles
Permanent flexion	Quadriceps

Hip.

Luxation	
Permanent flexion	Extensors of thigh
Permanent adduction	Glutei

Hand.

Flexion of fingers and wrist	Paralysis of all extensors
Extension of wrist	Flexors of forearm
Claw-hand	Extensors of forearm

Shoulder.

Subluxation	Deltoid

Trunk.

Dorsal scoliosis	Paralysis of one side of back muscles or other—erector spinæ group
Kyphosis	Extensor of back
Lordosis	Abdominal muscles

The development of deformity may be due to the assuming of a malposition, either to secure relief from discomfort and pain, or to maintain muscle balance. Curves are very likely to develop in the spine when a patient cannot maintain the erect posture because of muscle unbalance due to paralysis of the shoulder, back, or leg. Flexion deformities will develop in the leg if the patient is allowed to maintain the knee or hip in a flexed position. The use of pillows under the knees, a measure affording relief from pain, should never be allowed, since the maintenance of the knees in a flexed position will lead to flexion deformity.

As soon as the patient is ambulatory, *static influence* becomes a factor in the development of deformity. Weight-bearing aggravates slight malposition and not infrequently leads to the development of a bony deformity, which may require operative interference for correction. Weight-bearing and habit postures bear an important relation to paralytic deformity of the back and foot, and when the patient becomes ambulatory, one must be constantly on the alert for these influences.

The deformities due to the effect of *gravity* are the earliest to develop. They are seen especially in paralytic foot conditions. The most common is that of talipes equinus, which occurs in paralysis of the anterior muscles of the leg or in complete paralysis of the foot, when the force of gravity pulls the foot into the position of plantar flexion. The weight of the bed-clothes greatly aggravates the development of this deformity. Lack of proper protection of the limb when the patient sits up allows the foot to fall into equinus. Another deformity due to the force of gravity, but less commonly seen, is a dangling arm, which develops when the weight of the arm draws the humerus out of the glenoid cavity, in paralysis of the arm and shoulder muscles.

The Development of Deformity.—Deformities are progressive in character. The first change to occur is the contraction of the muscles, fascia, and ligaments on the unaffected side, and the relaxation and stretching of the muscles, fascia, and ligaments on the paralyzed side. At this stage, the limb may be forced easily into a correct position. If, however, the malposition is not corrected, deformity will increase, especially under the influence of weight-bearing and habit postures. In time, deformity becomes structural, and actual changes take place in the contour of the bones. Trophic changes occur in the bone from disuse and retarded circulation; such changes ultimately give rise to shortening. To allow deformity to develop to such a stage is inexcusable!

The Relation of Deformity to the Return of Muscle Power and Function.—The presence of deformity interferes with the return of muscle power. Recovery takes place more rapidly and more effectively in paralytic lesions when the joints of the paralyzed members are held in proper anatomical positions. The importance of preventing or of correcting a deformity cannot be overestimated.

Function is greatly lessened by the presence of deformity. Even if the improvement of muscle tone has been considerable, the function is impaired if deformity is present. After correction of the malposition, the improvement is often so great that weight-bearing is possible, even though muscle balance is not present.

The Prevention of Deformity.—The appreciation of the seriousness of a deformity, how it tends to increase, how it interferes with the return of muscle power, how it lessens function, and finally how it influences the life of an individual, should make it clear to us that the first step in the orthopedic treatment of poliomyelitis is the prevention of deformity. Malposition is obviated by maintaining the affected joints and limbs in correct anatomical position, until the time when functional control of the joint returns. or until its deficiency is compensated by mechanical or operative measures. The proper anatomical positions are as follows: The foot at a right angle to the leg, the hip in abduction, the spine hyperextended, the shoulder abducted, the elbow flexed to a right angle, the forearm supinated, and the wrist dorsiflexed.

Measures to prevent deformity should be carried out during the first few weeks of the disease, even before the exact location and extent of paralysis can be accurately determined. A fracture board placed under the mattress, and a hyperextension roll placed under the lower edge of the scapulæ will keep the spine in good anatomical position. The bedding should be kept away from the feet, as the weight of the bed-clothes will tend to cause plantar flexion, resulting eventually in contraction of the Achilles tendon and stretching of the anterior group of muscles. The feet should be kept at right angles to the legs by means of a padded box or other simple device.

In extensive paralysis involving both legs, it is best to immobilize the patient as soon as the condition warrants. Inasmuch as this immobilization is most efficient when complete, plaster affords a ready means to this end.* The plaster bandage is kept on day and night. It is split a few days after application to allow proper care of the skin and local therapy.

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* The technique of the application of a plaster bandage will be found in the Chapter covering Apparatus.

The objection that plaster causes atrophy has little significance. Rest in a plaster shell causes less atrophy than rest in bed in the position of deformity. Furthermore, any atrophy which may occur from disuse is easily overcome by massage and exercise.

In very severe cases it is necessary to wait until some of the involved parts have recovered before applying plaster or other means of immobilization. Meantime, the patient is kept in hyperextension with the feet at right angles and the hands tied to the top of the bed to keep the arms in abduction. Such a position affords comfort, prevents the development of deformity, and favors the recovery of muscle power.

As soon as the tenderness begins to disappear, usually in the course of about a week or ten days after the onset of the disease, it is possible to determine what muscles have been affected, and to take more definite measures to facilitate recovery of the involved part. In addition to immobilizing the involved limb in correct anatomical position, we should place it in a position of overcorrection, which not only will prevent deformity, but also will favor the return of muscle power by shortening the distance between the origin and the insertion of the weakened muscle. For example, if the inner muscle group of the foot is paralyzed, and all the other foot muscles are normal, the foot should be placed not only in anatomical position at a right angle to the leg, but it should be placed also in varus, a position that will favor the return of strength in the affected muscle group. This principle of overcorrection is a most important factor in the treatment of poliomyelitis. It is particularly applicable in the first six weeks of paralysis, the time when the greatest return of muscle power takes place.

Careful supervision should be continued when the child is ambulatory. Much harm may be done in allowing the patient to sit up or bear weight with a limb held in an incorrect position. For instance, the force of the body-weight in mild talipes valgus serves only to aggravate the deformity. Under the influence of weight-bearing, shortening of the leg will give rise to scoliosis, even though the spinal muscles are not involved. In spinal involvement, the danger of the development of deformities in the ambulatory stage is particularly great. A curvature may develop insidiously, and may not be recognized until a definite deformity is present. Once the curvature has started, it is aggravated by the patient's assuming the erect position, since all the influencing factors unilateral muscle power, contractures, and bad posture due to weakness and to the effort to protect the paralyzed muscles—are present.

During the ambulatory stage, some form of apparatus should be worn to prevent the development of deformity. If the balance is good, but a general muscle weakness persists, a light protective apparatus allows the weakened part to rest, thereby preventing muscle fatigue, which would be likely to cause poor posture and eventually terminate in deformity. If, when the patient is ambulatory, the muscle balance is not good because of a persisting unopposed muscle pull, apparatus must be worn to maintain the involved part in overcorrection.

In involvement of the back muscles, a brace or close-fitting corset lessens fatigue and prevents the development of a malposition. If the abdominal muscles are paralyzed, a corset prevents muscle stretching and the development of secondary deformity of the lower chest. In paralysis of the lower extremities, particular care must be exercised in providing the limbs with proper support, so that no deformity will occur when the patient sits up or bears weight. When the shoulder muscles are involved, apparatus must be worn to keep the shoulder in abduction; otherwise, the weight of the arm and the pull of gravity will stretch the shoulder muscles. In wrist paralysis, support is necessary to maintain the hand in hyperextension.

Some objections have been raised to the use of apparatus. The only potent argument against it is that apparatus, which is applied to rest the limb and to prevent deformity, occasionally does not accomplish this purpose. The objection that limbs become atrophied when apparatus is worn is of little importance, since any wasting can be overcome by proper therapeutic measures, such as massage and exercise.

The Correction of Deformity.—We have spoken in detail of the importance of the prevention of deformity. In cases in which deformity occurs from lack of proper treatment, its correction is imperative. There is no period in the course of the disease when the correction of deformity, whether of tissue or bony variety, is not indicated. If this fact were appreciated, much of the disability resulting from deformity would be avoided. If we stop to consider the sequelæ that result from an original deformity, which may be only a very slight one, we realize the importance of correction in the very early period. For instance, a slight flexion of the knee will interfere with the return of power in the quadriceps group. If the patient is allowed to bear weight with the knee flexed, the disturbance in statics will give rise to the development of secondary deformity in the form of a slight knock-knee. Another illustration, showing the development of a series of secondary deformities, is seen in the contracture of the knee, the tightening of the ilio-femoral band, and finally scoliosis, sequelæ that are found associated with an old-standing toe-drop. It is most unfortunate that such deformities are allowed to develop. If care were taken to prevent the original deformity, or to effect its correction immediately upon recognition in the early stages, the sequelæ would be avoided.

A detailed study of the deformities that develop in the different parts of the body, as well as the most satisfactory means of preventing and correcting them, will be found under the treatment in the Late Paralytic Stage and under the specific treatment of each body part.

Conclusions.—The prevention of deformity and the correction of deformity as soon as recognized are the most important factors in the treatment of infantile paralysis. Since we have a complete knowledge of the etiological factors that enter into the formation of deformity, and have means at our disposal to prevent and correct malposition, few cases of structural deformity should ever develop. We must be on the alert for the occurrence of deformity at all times. As soon as recognized, at whatever stage of paralysis, it should be corrected. Mechanical means, manipulative measures, and operative treatment should be used as necessary.

The Restoration of Muscle Power.—A complete description of the forms of conservative measures used in the restoration of muscle power will be considered in a separate chapter.

TREATMENT IN THE LATE PARALYTIC STAGE.

In the late stage of poliomyelitis, the paralysis is stationary, and any deformities are more or less fixed. This period may be considered as beginning one and a half to two years after the acute attack. Improvement under conservative treatment, for the most part, has ceased. The child is usually wearing some type of protective apparatus as a means of preventing deformity, or as an aid to locomotion. It is well to continue massage, exercise, and hydrotherapy in the late paralytic stage, for such treatment helps to diminish muscle atrophy by maintaining good circulation. Functional use of the affected part, however, is of more value than therapeutic measures, and should be encouraged.

The treatment of paralysis during the late stage is often surgical. Due to the great advance that has been made in the operative treatment of paralysis, it is possible, by surgical procedures, to restore power and to increase function in many limbs which, under the earlier forms of treatment, would never have regained functional use.

Operative measures are rarely used until two years have elapsed from the initial attack, since during that period, improvement may be expected under conservative treatment. Generally, operative interference should be postponed until the child is seven or eight years old, for surgical procedures performed before this age may prove to be unnecessary or not adapted to the problem when the child reaches adolescence. Exceptional cases, requiring operation before the lapse of two years and before the child has reached the age of seven years, are those in which it has not been possible to correct deformity or contractures by conservative means. In such cases, operative procedures are indicated at any stage, even if their value is only temporary in maintaining the foot in good position until further operative interference is advisable.

Operative treatment is undertaken for two purposes: (1) *To correct deformity*, and (2) *to improve function*. A rule applicable to practically all joint surgery is that deformity, which disturbs the joint equilibrium, should be corrected before surgical measures are used to improve function. The operative procedure used to correct deformity must be carefully selected by the surgeon. In general, a manipulative or stretching process is preferable to an open operation, even though it is necessary to repeat the manipulative procedure several times before correction is effected. While it is very simple to secure correction of a tendon contraction by tenotomy, the contracture of other structures about the joint is not overcome in the process, and deformity frequently results. Tenotomy tends to weaken a muscle group, whereas manipulation or stretching in no way impairs its power.

Mild tissue contraction or slight bony deformity may usually be overcome by forcible manipulation. In exceptional cases in which it may be found necessary to use more drastic measures, manipulation may be supplemented by tenotomy, tendon lengthening or shortening, or fasciotomy. In the most severe cases, the bones are so misshapened that it is necessary to attack the bony structures to correct deformity.

Among the operations that have for their purpose the improvement of function are those directed to the restoration of muscle balance, and those designed to secure stabilization of a joint. The choice of operative procedure depends upon the extent of the paralysis in a given area. The selection often hinges upon the judgment and experience of the surgeon, who chooses the measure that seems particularly adaptable to the case at hand.

In selected cases, tendon transplantations give good results.

Their use in general, however, has not been sufficiently satisfactory to warrant their popularity in the last few years.

Nerve grafting and transplantations have shown no permanent results.

When all the muscles in a given area are paralyzed, and there is not only loss of voluntary power, but also relaxation of the joints, stabilizing procedures are indicated. Numerous operative procedures have been devised to stabilize the joint, and among them are: Astragalectomy, arthrodesis, artificial silk ligaments to reinforce the joints, tenodesis, and other plastic operations. These procedures will be taken up in detail under the Operative Section.

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CHAPTER X.

TREATMENT OF THE LOWER EXTREMITIES.

PARALYTIC INVOLVEMENT OF THE FOOT.

The Development of Instability and Deformity.—The normal foot is balanced by muscles that work in perfect coördination, assisted by ligaments which check the extremes of motion. When complete paralysis involves all the muscles of the foot, the ligaments become lax, and the foot is correspondingly flail. Deformities are not likely to develop in such cases.

Total loss of power, however, is rare. Even in extensive involvement, slight power usually remains in the plantar flexors of the toes, as evidenced by the development of cavus deformities during convalescence. Paralysis of single muscle groups is the more common involvement. In such cases, the opponents of the paralyzed muscles, which are normal and active, exert a pull upon the weakened muscles, and eventually will give rise to the development of deformity, unless preventive measures are undertaken.

The importance of recognizing an unopposed muscle pull, and thereby anticipating the development of deformity cannot be overestimated. It is likewise important to correct deformity as soon as it is recognized, regardless of the stage of the disease.

Pathological Changes.—Early in paralysis, the ligaments about the ankle-joint relax. When the strong muscles in the foot begin to pull on their paralyzed antagonists, the ligaments in the region of the active muscles contract, thereby aggravating the development of instability and deformity. The contraction of the ligaments may be easily reduced, provided deformity is corrected early in the convalescent stage, but if the malposition is allowed to continue, the ligaments may offer some difficulty when reduction is attempted.

In long-standing deformity the normal relations of the articular surfaces of the bones of the foot are altered. The joint and bone alterations consist mainly of atrophy, absorption of lime salts, and thinning of the cartilage. The greatest changes take place at the mid-tarsal joint and in the astragalus.

Diagnosis.—The determination of paralytic involvement of the foot should be made early in the attack, so that the development of deformity may be anticipated, and steps taken to prevent it.

The diagnosis of paralytic involvement of the foot is easily and satisfactorily made by testing the power of the muscles that control the four principal movements of the foot, namely, dorsiflexion, inversion, eversion, and plantar flexion. Dorsiflexion is accomplished by the anterior tibial muscle and the dorsiflexors of the toes; inversion is accomplished by the anterior and posterior tibial muscles; eversion by the peroneal muscles; and plantar flexion by the gastrocnemius and posterior tibial muscle.

The examiner, in making the tests, holds his finger a few inches away from the extremity, and requests the patient to invert, evert, or flex his foot. If the various muscle groups are present, the patient will be able to move the extremity toward the examiner's finger. In a young child the muscles may be tested by pricking the different sides of the foot with a pin; if the muscle group is present, the child will suddenly turn his foot away from the pin, but if absent, he will turn his foot toward the pin.

If deformity is present, its cause may be traced to paralysis of certain muscle groups. In talipes equinus, the anterior tibial muscle is paralyzed or weakened; in talipes valgus, the anterior and posterior tibial muscles are affected; in talipes varus, the peroneal muscles are weak; and in talipes calcaneus, the gastrocnemius is involved.

In many cases the posture that the patient assumes in walking, as well as the wear on the shoe, will aid in making the diagnosis.

Prognosis. The prognosis of paralysis involving the foot, as the prognosis of paralysis of any body part, depends upon the extent of the cord lesion. As a general rule, the initial involvement is more extensive than the residual paralysis, and some improvement may be expected within the first two weeks to six months after the onset of the attack. If, at the end of six months, the condition is one of complete or nearly complete paralysis of all the muscles involving the foot, then recovery of muscle control is not possible, and the limb must be stabilized by operative procedures. If power is only partly lost, stabilizing operations, and in selected cases, tendon transplantations, offer means of securing a useful foot. If the muscles have been stretched as the result of deformity, they may recover entirely, or at least improve, upon the correction of the deformity followed by appropriate local and general treatment.

Deformity, of course, should be prevented. If, however, distortion has occurred from lack of protective measures, it should be corrected as soon as recognized. Many cases of deformity can be successfully overcome by conservative procedures. If these are not sufficient, the use of operative measures will give the patient a good weight-bearing ankle and foot.

Treatment.—When paralysis involves the foot, measures should be taken at once to avoid the development of deformity, by placing the foot in proper anatomical position, that is, at a right angle to the leg. Plaster or some form of splintage may be used to maintain the foot in this position. As soon as it is possible to determine the muscles affected, the foot should be held in an overcorrected position to prevent contraction of the strong antagonists and to favor the regeneration of the weakened muscles. Daily manipulation of the foot into an overcorrected position also should be practised. When the deformity is not reducible by these measures, a forcible manipulation and stretching under ether should be done, followed by the application of a plaster bandage. In certain cases it may be necessary to perform a tenotomy or tendon lengthening. When deformity has progressed to the stage where the bones are distorted and the joints are lax, it is necessary to reëstablish anatomical relations by surgical means. Stabilizing operations are very successful, and are among our most useful procedures in dealing with instability involving the foot and ankle.

TALIPES VALGUS.

Talipes valgus is characterized by abduction of the foot at the mid-tarsal joint, by elevation of the outer border of the foot, by depression of the long arch, and by contraction of the toes (Fig. 8). The degree of deformity varies according to the severity of the paralysis. In mild cases the abduction is slight, and the arch is depressed only upon weight-bearing. When the muscle involvement is severe, the abduction at the mid-tarsal joint may amount to 15 degrees or more, and the arch is permanently flattened.

Etiology and Pathology.—Talipes valgus of paralytic origin is due primarily to involvement of the inner group of muscles of the lower leg. The muscles that supinate the foot are the tibiales anticus and posticus, the extensor longus hallucis, and the flexor longus hallucis. The most important of the supinators is the tibialis anticus, and it is this muscle that is paralyzed most frequently. Its involvement alone, or in combination with paralysis of the tibialis posticus or other supinators of the foot, gives rise to valgus deformity. The extensor longus hallucis and the flexor longus hallucis, which have only a slight supinating action, play a less important part in the production of a talipes valgus.

Paralysis of the inner muscle group may persist for some time before any anatomical changes take place. Gradually, the astragalo-scaphoid and the calcaneo-astragaloid ligaments relax and stretch. Then the opposing muscles, in particular the peronei, contract, and exert an unopposed pull on the paralyzed muscles and the relaxed ligaments. Eventually the foot becomes fixed in the position of valgus. The deformity may develop while the patient is still recumbent, but it often does not take place until weight is borne on the foot. Mild contraction of the Achilles tendon, due to the contraction of the strong posterior muscle group, is frequently found associated with a valgus deformity.

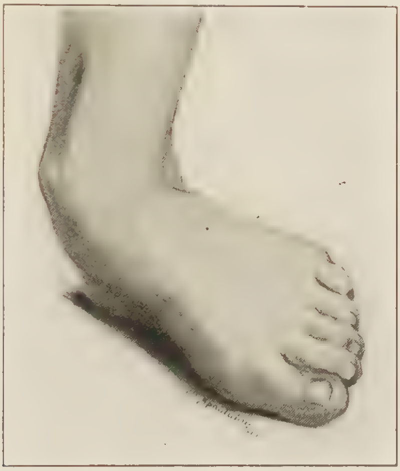

Fig. 8.—Talipes valgus.

Weight-bearing, in the presence of even a slight deformity, tends to aggravate the condition. The scaphoid sags, the astragalus rotates, and abduction takes place at the mid-tarsal joint. The scaphoid may continue to sag until it finally rests on the floor. The abduction at the mid-tarsal joint often amounts to 15 degrees or more, and in long-standing cases, eversion of the lower leg is found associated with it. This eversion in turn influences the development of shortening. There is usually some rotation of the os calcis at the calcaneo-astragaloid joint, which is seen clearly when the foot is viewed posteriorly. In time, the changes become so marked that deformity resists correction, and the limb is practically useless.

Symptoms.—Patients consult the orthopedic surgeon for limp and deformity. The limp is due primarily to muscle weakness, and it is greatly aggravated by the loss of muscle balance and the presence of deformity and shortening. Instability is very marked when eversion of the lower leg is associated with abduction at the midtarsal joint; in walking, the foot drops, and the patient trips unless he lifts the leg high before placing the foot on the floor.

Diagnosis.—The determination of a paralytic talipes valgus may be made by a muscle test. The examiner, holding his finger to the inner side of the patient's foot, requests him to invert it. If the inner group of muscles is paralyzed, the patient will be unable to do so. In young children, the test may be made by pricking the outer side of the foot with a sharp point. If the inner group of muscles is present, the child will suddenly invert his foot, but if the muscles are weak or absent, he will evert his foot toward the point. It is often difficult to determine the degree of paralysis in the young child, but fortunately such a diagnosis is rarely necessary, since recognition of the muscles involved is sufficient to enable us to take steps to aid their recovery and to prevent the development of a deformity.

Paralytic talipes valgus should be differentiated from talipes valgus of congenital origin. The presence of an extreme valgus deformity of one foot is suggestive of paralysis; congenital talipes valgus always involves both feet. Further means of differentiating the two conditions are the lack of muscle atrophy and the presence of muscle control in cases of congenital origin.

Prognosis.—The prognosis of paralytic valgus is dependent upon the extent of the paralysis in the inner group of foot muscles, and upon the degree of the deformity. When only a slight weakness or deformity is present, a good functional result is ensured by the use of conservative therapeutic measures. When the paralytic involvement is extensive, the forces producing deformity have free play, and it may be impossible to hold them in check by conservative treatment. In such cases, surgical measures are usually indicated to correct the deformity and to stabilize the foot.

Treatment.—PREVENTIVE.—Before we consider the treatment of a talipes valgus, let us discuss the possibility of the prevention of such deformity. Since it is possible to determine paralysis of the inner group of muscles early in the attack, the deformity of talipes valgus may be anticipated while the patient is still recumbent. In order to counteract the pull of the strong outer group of muscles and to prevent the weakened muscles and relaxed ligaments from becoming stretched, the foot should be placed in a light plaster bandage or in some form of splintage, in the position of slight varus and at a right angle to the leg. If the etiology of the deformity were appreciated, and precautionary measures taken to avoid distortion in all cases, there would be few cases of structural valgus deformity, and there would be little necessity for the use of operative measures.

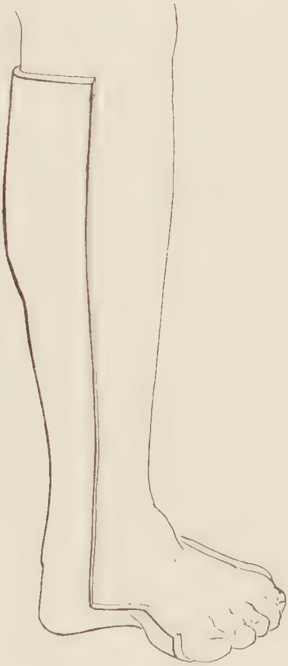

FIG. 9.—Talipes valgus. Foot in a bivalved plaster bandage, at a right angle to the leg and in varus.

CONSERVATIVE.—If a slight talipes valgus develops in the early stages, the same measures that are undertaken to prevent the deformity should be carried out, that is, the foot should be placed at a right angle and in slight varus in a light plaster bandage or in some form of retentive apparatus, extending from the knee to the toe (Fig. 9). Within a few days the plaster is bivalved, and local therapy started. The bandage remains on night and day and is removed only during local treatment.* The foot should be manipulated into varus by the attendant, fifty times each morning and night, to prevent any tendency to contraction of the unaffected groups (Fig. 10). The above treatment is carried out until supportive measures are indicated, or until recovery has taken place.

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* A detailed description of the forms of local treatment will be found in the Chapter on the Restoration of Muscle Power.

As soon as weight-bearing begins, instruction in walking is advisable. Most individuals have a tendency to walk with the foot everted, a position that favors the development of deformity. The patient should be taught to walk with the feet straight ahead.

Mechanical.—When the patient becomes ambulatory, usually in the course of three or four weeks, a new factor—body-weight—must be carefully considered, as it bears an important relationship to the development of deformity. The strain of the body-weight will cause any existing deformity to increase, unless the foot is properly protected. When the patient begins to walk, the foot is usually held in the plaster bandage, which at this stage affords sufficient support. Upon the removal of the bandage, some form of mechanical apparatus is often necessary to protect the foot against the development of structural deformity.

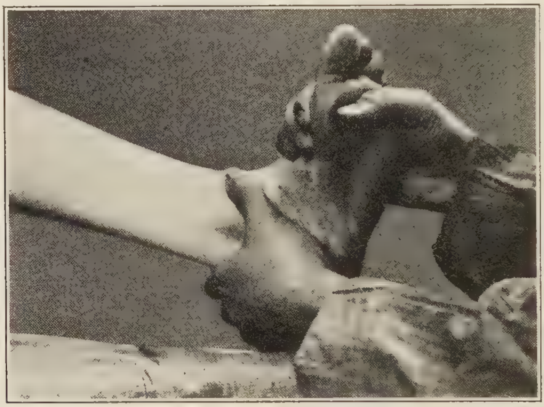

Fig. 10.—Manipulation of the foot into varus.

The types of apparatus selected for this purpose will depend upon the severity of the case and upon the judgment of the surgeon.* When there is only a slight weakness, a raised heel and sole is sufficient to maintain the foot in proper position. If there is a distinct tendency to pronation, a Thomas heel or a Whitman brace, or a combination of both, is more efficient. A valgus shoe with raised heel and sole may also be used in slight deformity. If advisable, a

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* For complete description of the various braces, see Chapter XIV, Mechanical Section.

Whitman brace may be used in conjunction with it. If the toes are contracted, an anterior arch support is worn.

In the more severe case, a valgus brace, by means of which any degree of overcorrection is obtainable, should be worn. If patients object to wearing it in the daytime because it is cumbersome, some other form of apparatus may be worn during the day, and the valgus brace applied only at night.

Operative.*—When a talipes valgus develops in spite of conservative treatment, operative interference is required to correct the deformity and to maintain the limb in proper position. Such interference is justifiable at any stage of convalescence.

The operative procedure selected to correct the deformity depends upon the degree of disability, the severity of the deformity, and the function of the muscle groups present.

Manipulation.—Forcible manipulation and stretching of the foot, either by hand or with the aid of a Thomas wrench, is often effective in overcoming a contracture of the outer and posterior muscle groups. The procedure should be carried out with the patient under ether. Following manipulation, the foot is maintained in the position of varus and slight calcaneus. If the first manipulation is unsuccessful, the process may be repeated several times.

Tendon Transplantation.—Occasionally transplantation of the peroneal tendons into the scaphoid may be used to correct a valgus deformity, but the power of this group can rarely be substituted for that of the strong inner group of muscles. Transplantation of the peronei into the scaphoid is more effective when combined with an astragalo-scaphoid arthrodesis.

Tenodesis.—This procedure is of most value when used in conjunction with an astragalo-scaphoid arthrodesis. Occasionally, however, it may be used satisfactorily alone.

Astragalo-scaphoid Arthrodesis.—Mild deformity can be overcome by arthrodesis of the astragalo-scaphoid joint. Transplantation of one or both peronei into the scaphoid, or fixation of the anterior tibial muscle into the tibia, may be done as supplementary measures to the astragalo-scaphoid arthrodesis in cases in which the arthrodesis is not efficient in controlling the mechanical problem. An astragalo-scaphoid arthrodesis, with or without such supplementary measures, may be performed even before the two-year convalescent period has elapsed. In young children it may afford only temporary relief, but it has the advantage of holding the deformity in check until the patient is at the age when operations of a more radical nature may be performed with safety. In children between the ages of eight and twelve years, who have a simple valgus deformity, correction can be secured by an astragalo-scaphoid arthrodesis.

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* For details of operative technique, see Chapter XIII, Operative Treatment.

Wedge Osteotomy.—In severe and long-standing cases of talipes valgus, in which the deformity is fixed, and in which, as a rule, more than one muscle group is affected, it is often necessary to take a wedge out of the astragalo-scaphoid region to correct the deformity. In some cases, the correction may be made at the time a stabilizing operation is done.

Astragalectomy and Subastragalar Arthrodesis.—Stabilizing operations are often necessary to correct severe talipes valgus. They are always required when the paralysis of the anterior leg muscles is combined with weakness of other muscle groups. Astragalectomy should not be performed until the child is at least eight years of age, and a subastragalar arthrodesis is not advisable before the age of twelve years. Both operations should be withheld until two years have elapsed from the onset of the disease. Deformity should always be corrected before stabilizing measures are carried out.

It depends upon the age of the patient and upon the mechanical problem confronted whether an astragalectomy or a subastragalar arthrodesis is advisable. When the child is between the ages of eight and fifteen years, an astragalectomy may be performed with the assurance of a successful outcome. A subastragalar arthrodesis is the operation of choice when the patient is between the ages of fifteen and twenty years, and may be advisable at an earlier age in cases of general weakness of all the foot muscles, providing there is good stability at the astragalo-tibial joint.

TALIPES VARUS.

In talipes varus, the outer side of the foot sags and the forefoot inverts at the mid-tarsal joint (Figs. 11 and 12). Little or no power of eversion remains. This deformity is often seen in combination with other deformities, most frequently with talipes equinus.

Etiology and Pathology.—Varus may develop when the peroneal muscles are partly or completely paralyzed, or it may occur in total paralysis of all muscle groups of the foot. The muscles on the inner side of the foot, when unopposed, contract, and in so doing stretch the peronei. The plantar fascia and ligaments on the inner side of the foot, particularly the astragalo-scaphoid ligaments, also contract and hold the foot firmly in the position of varus, thus paving the way for the development of a structural deformity. Frequently, the Achilles tendon becomes slightly contracted, giving rise to a mild talipes equinus.

The first bony change to take place is a twist in the neck of the astragalus. Displacement occurs between the astragalus and the midtarsal joint. As the deformity becomes fixed, the cuboid stands out prominently. In extreme deformity the os calcis rotates inward (Fig. 13). The longer such bony changes are allowed to continue, the more they increase and the more difficult they are to overcome. In long-standing cases, objectionable callous formations and bursæ develop under the cuboid.

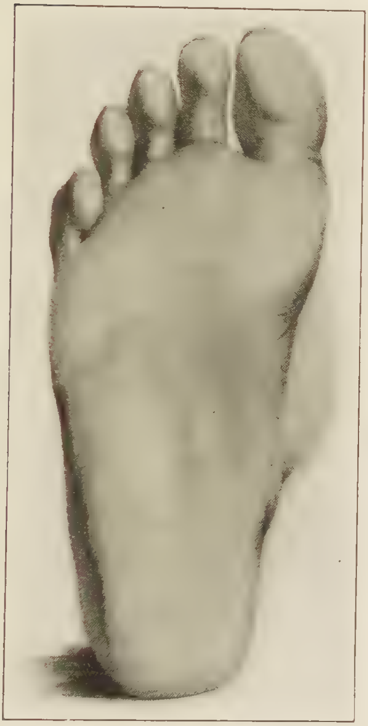

Fig. 11.—Normal sole.

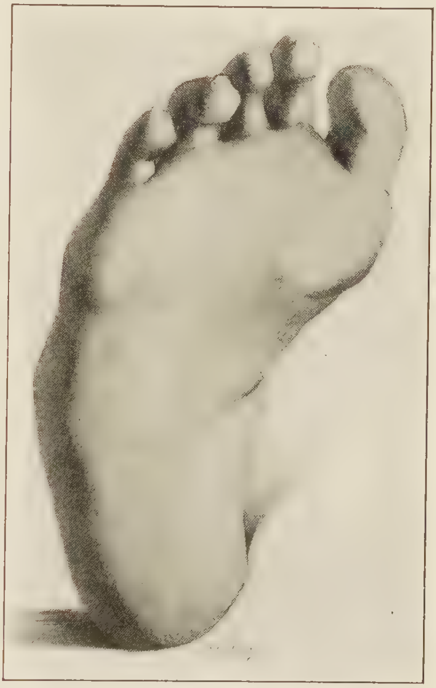

Fig. 12.—Varus sole.

Symptoms.—Limp and deformity are the common complaints. The unequal muscle balance between the inner and outer muscle group aggravates the limp. If the varus deformity is severe, shortening may be present as the result of disuse.

Diagnosis.—The absence of the peroneal group is determined by a muscle test. If the patient cannot evert his foot toward the examiner's finger, which is held well to the outer side of the foot, the power in the peroneal muscle group is very weak or completely lost. In old-standing cases, the limp, the supinated and adducted position of the foot, the atrophy of the calf muscle, and shortening are pathognomonic of paralytic varus.

At first glance, paralytic talipes varus bears a close resemblance to congenital varus. Examination shows that in the paralytic type there is usually more equinus deformity, but less supination and cavus. The deformity of congenital origin involves the entire tarsus; the deformity of paralytic origin is due principally to subluxation of the joints. Another characteristic peculiar to the paralytic cases is the contraction of the toes. In congenital cases the toes are usually in normal relation, although in long-standing cases pathological changes may have occurred.

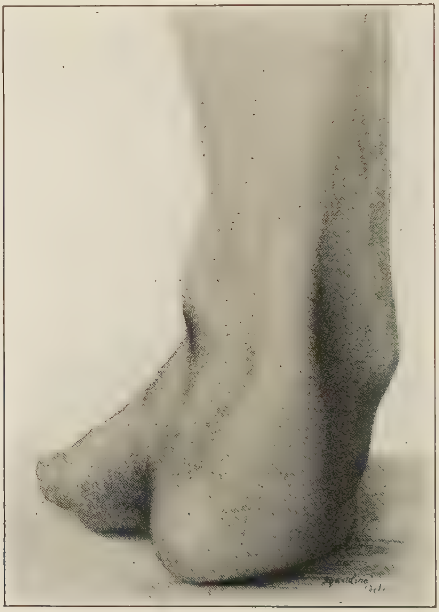

FIG. 13.—Talipes varus, showing inward rotation of the os calcis.

The presence of shortening, atrophy, and coldness of the limb will also help to distinguish the paralytic cases.

Prognosis.—The prognosis in simple varus deformity is good, both as to the correction of the deformity and as to the return of function. Simple cases may be treated by conservative measures. When deformity cannot be held in check by conservative treatment, operative measures are efficacious in correcting the deformity and in reëstablishing function. When there is a general weakness of all muscle groups in addition to the involvement of the peronei, a good weight-bearing foot may be secured by stabilizing operations.

Treatment.—PREVENTIVE.—The deformity of talipes varus may be prevented by carrying out proper treatment immediately upon recognition of the weakness of the peroneal muscle group. The foot should be maintained in a position favorable to the return of power in the weakened muscles, by means of a light plaster bandage, extending from the toes to the knee with the foot at a right angle to the leg and in slight valgus.

CONSERVATIVE.—If a varus deformity develops during convalescence, the weakened extremity should be placed in a plaster bandage as described above. Upon the disappearance of tenderness, usually in about ten days or two weeks, the plaster is bivalved to permit local therapy.* Except for the removal of the plaster bandage during the period of treatment it should remain on day and night.

Manipulation of the foot into valgus is an important measure, and as soon as the acute symptoms have subsided it should be carefully carried out by the attendant each morning and night.

In four to six weeks, provided there are no complications, the patient is allowed to walk on the plaster bandage, the sole of which has been reinforced. At this time the patient should be taught to walk with the foot straight ahead to avoid the development of habit limp and contractures.

The plaster is usually removed in the course of ten weeks. If muscle recovery has not taken place within this time, some form of apparatus will be required to prevent recurrence of the deformity.

MECHANICAL TREATMENT.†—The selection of the form of support depends upon the severity of the lesion. In mild deformity the raising of the sole on the outer side of the shoe will maintain correction. When the deformity is slightly obstinate, the varus shoe may be used to maintain the foot in valgus. This form of support is especially effective when combined with a lift on the outer heel and sole.

In severe cases, a varus brace, by which it is possible to obtain any degree of correction, is advisable.

OPERATIVE TREATMENT.‡—If the deformity cannot be corrected or held in check by conservative measures, operative interference is indicated. It may be carried out at any stage of the disease, even if two years have not elapsed from the onset of paralysis. The operative procedure selected will depend upon the severity of the deformity and the function of the muscle groups present.

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* The forms of restorative treatment and appropriate exercises will be found outlined in the Chapter on the Restoration of Muscle Power.

† A complete description of the various types of apparatus will be found in Chapter XIV on Mechanical Treatment.

‡ For detailed description of the technique, the reader is referred to Chapter XIII on Operative Treatment.

Manipulation.—Forcible manipulation and stretching under ether may be undertaken at any time during the course of treatment. It is a safe procedure and may be repeated several times. After manipulation, the foot should be maintained in a plaster bandage in the position of valgus and slight calcaneus.

Wedge Osteotomy.—This procedure is used when repeated manipulations have failed to correct the deformity, or when a bony deformity exists in the astragalus. A V-shaped piece of bone of sufficient size to permit the deformity to be overcorrected is removed from the outer side of the astragalus. After the removal of the wedge, the foot is held well overcorrected, pending a transplantation.

Transplantation of the Anterior Tibial Tendon into the Cuboid.—The results of this operation are very satisfactory, for it reëstablishes the power of eversion and dorsiflexion.

As a rule, the child should be at least eight years of age before such a transplantation is done, but it may be used earlier in exceptional cases when it is impossible to prevent the development of the deformity by conservative means. A transplantation may be combined with an operation for the correction of a fixed varus deformity, or it may be done a few months after deformity has been corrected. The writer has found that the best results are obtained when the latter course is followed.

Gallie Operation.—This operation may be used in cases in which bony deformity does not exist, and in which a tendon transplantation would not be sufficient to maintain correction of deformity. In this operation, however, it is difficult to secure the right fulcral point for fixation, and if considerable tension is necessary to maintain correction, stretching occasionally takes place with a consequent recurrence of slight varus deformity. The Gallie operation has definite value when performed in conjunction with a stabilizing measure.

Astragalectomy and Subastragalar Arthrodesis.—When there is a general involvement of the foot muscles in addition to total paralysis of the peroneal group, a stabilizing operation is indicated. Whether an astraglectomy or a subastragalar arthrodesis is advisable depends upon the age of the patient and the condition of the foot muscles.

TALIPES EQUINUS.

The most common deformity resulting from an attack of poliomyelitis is talipes equinus, or toe-drop. It may develop when the lower leg is totally paralyzed; it may be static in origin, occurring as the result of shortening of the leg; and finally, it may be due to paralysis or weakness of the anterior muscles of the lower leg (Fig. 14). In this section we are concerned with equinus due to the last condition.

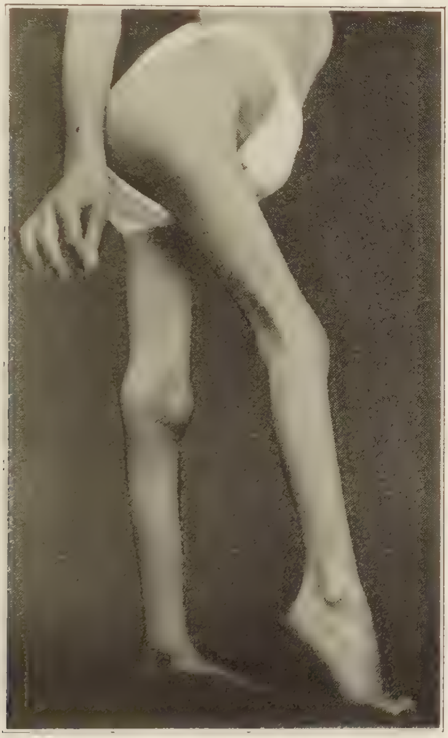

FIG. 14.—Talipes equinus, showing secondary deformities of contracted knee, contracted iliopsoas, and lordosis.

Etiology and Pathology.—The anterior muscles may be wholly or partly paralyzed, depending upon the extent of the damage to the motor cells governing the anterior tibial nerve. In some cases the tibialis anticus and the extensor digitorum are affected, but the extensor hallucis remains normal. In other cases only one muscle may be involved.

The severity of the deformity is frequently out of proportion to the existing pathology. This is because certain muscles, which were not originally involved in paralysis, or which were only slightly weakened, become stretched when an equinus deformity is allowed to develop. Examination of these muscles shows them to be still pink in color, indicating that they are stretched and not paralyzed. It is in these muscles that regeneration takes place when deformity has been corrected.

Once paralysis or weakness of the anterior muscles occurs, the force of gravity causes the forefoot to sag. The bed-clothes pressing on the foot will weigh it down even more. If the foot is allowed to remain in this position for any length of time, the posterior group of muscles, whose action is unopposed, gradually contracts, and the heel is slowly pulled upward and backward. In time, such contractions become fixed, and bony changes take place. The astragalus may tilt downward and backward until it articulates with the posterior surface of the tibia. In such extreme deformities, the cartilage disappears from the part of the astragalus that is no longer in contact with the tibia and fibula.

Weight-bearing aggravates the deformity, and skeletal changes take place more rapidly when the patient is ambulatory. Very tender callous formations develop under the flattened anterior arch. In extreme and long-standing cases, the foot may be turned directly backward. A case of this kind came to the attention of the writer, in which the deformity was so extreme that the patient bore weight on the dorsum of the foot.

Talipes equinus is often complicated by other deformities. Talipes varus or valgus may develop if the patient bears weight with the foot in the position of equinus. Cavus, due to contraction of the plantar fascia, is the most common complication. If the thigh muscles are paralyzed, recurvatum of the knee may develop (Fig. 15). It is also not uncommon to find contraction of the knee-joint and of the hip, since a talipes equinus apparently lengthens the leg and makes it necessary for the patient to flex his knee and hip in walking. In such cases, scoliosis or lordosis may develop as secondary deformities.

Symptoms.—The surgeon is consulted because of increasing limp and disability. The patient usually walks on the ball of the foot and in extreme cases on the dorsum of the foot.

If the quadriceps is weak, steppage gait is always present, and the patient lifts the knee high in order to avoid tripping.

Diagnosis.—The presence of toe-drop, the loss of power in whole or in part in the anterior group (Fig. 45), and the contraction of the posterior muscle group enable one to make a diagnosis.

In old-standing cases, actual shortening is always present, in spite of the fact that the leg apparently is longer.

Occasionally, it is necessary to differentiate paralytic talipes equinus from spastic equinus. In spastic paralysis, the deformity is more rigid, all the muscles are functional, the knee-jerk is increased, and a similar condition of spasticity often exists in the arm or in the other leg.

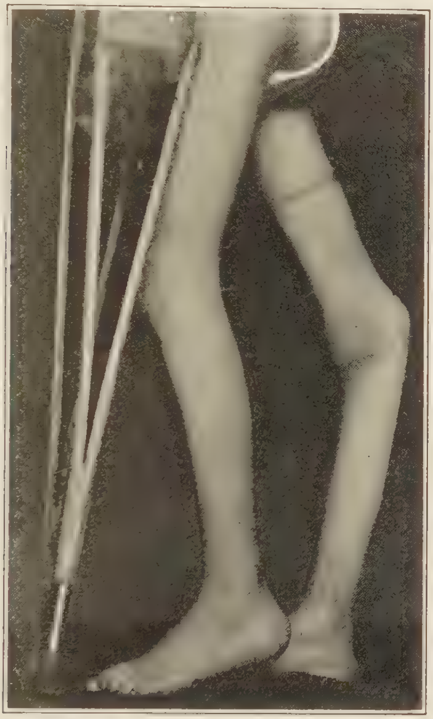

Fig. 15.—Genu recurvation of the right leg, occurring when talipes equinus is associated with thigh paralysis.

Prognosis.—The outcome of a talipes equinus depends, of course, upon its early recognition. It is possible to prevent or to correct contractures in the early stages. If the deformity has existed for some time, manipulative or operative treatment is necessary to secure correction and make the foot functional. In such cases, the muscle recovery is very slow.

Treatment.—Preventive.—The development of a talipes equinus deformity may be avoided by applying some form of apparatus or a plaster bandage as soon as the weakness in the anterior muscle group is recognized.

In the very early paralytic stage, padded boxes will support the foot at a right angle to the leg, and a cradle will prevent the bedclothes from pressing on the toes. When tenderness has subsided sufficiently, a plaster bandage, applied from the toe to just below the knee with the foot in 10 degrees dorsiflexion, is efficient in holding the foot in overcorrection. This position favors the return of muscle power and prevents the development of a posterior contracture. After a period of two or three weeks, during which time the foot is at absolute rest, the plaster is bivalved, and the anterior half removed to permit local therapy. Manipulation of the foot into dorsiflexion should be carried out fifty times each morning and night by the attendant, to assist in preventing any contraction of the Achilles tendon.

In about six weeks from the onset of the attack, the patient may walk on the plaster bandage.

Conservative.—If an equinus deformity develops during convalescence, it often may be overcome by the application of a plaster bandage, as described above. In some instances, the following method is effective in correcting a contraction of the Achilles tendon.

A plaster bandage is applied from the toes to just below the knee, with the foot in as much dorsal flexion as possible. Successive wedges are then removed from the anterior part of the plaster. By means of straps and buckles fastened around the foot and attached to the top of the bandage, continued tension is made on the foot to pull it into dorsal flexion. The deformity, if slight, often may be corrected in a week by this method; older cases may require two or three weeks.

Mechanical Treatment.*—A posterior wire splint may be used in the early stage of talipes equinus in place of a posterior plaster shell. An arthrodesis brace holding the foot in 5 degrees dorsiflexion may be used when the patient bears weight. Care must be taken that the straps on this brace are not constricting.

When the foot may be actively exercised, the equinus brace or the equinus shoe, both of which are equipped with stop-joints to prevent plantar flexion, may be used.

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* For details, see Chapter XIV—Mechanical Treatment.

OPERATIVE TREATMENT.*—Upon the failure of conservative means to hold the deformity in check, operative interference becomes necessary. Surgical measures are advisable in advanced cases to facilitate recovery and to shorten the period of convalescence. The type of operation indicated in a given case depends upon the duration and rigidity of the deformity.

Manipulation and Stretching of the Achilles Tendon.—This procedure corrects mild contractures very satisfactorily. If the deformity is of old-standing, an Achilles stretcher is helpful in increasing leverage at the time of the manipulative process. The manipulation is followed by the application of a plaster bandage to hold the foot in an overcorrected position. Later, this plaster is split, but the posterior shell remains on until correction is assured, or until the condition permits the use of a brace.

Manipulation may also be of value in correcting extreme deformity, in which extensive bone operations are contraindicated because they might lead to disturbance of the general nutrition and circulation of the foot.

The following case is an interesting illustration of the correction of an extreme deformity by repeated manipulations. A good result with perfect motion was obtained. The case is also of interest in that power returned to the anterior muscle group, when the deformity, which was of many years' standing, had been corrected.

CASE I.—R.R., aged twenty-two years.

Complaint: Deformity of the left foot.

Duration: From infancy.

Cause: Illness in early childhood, undoubtedly infantile paralysis. The trouble with the foot dated from this time.

Physical Examination: Left foot in the position of equinovarus, so extreme that on standing the dorsum of the foot bore the body weight. A large callosity covered the dorsum of the foot. The Achilles tendon and the plantar fascia were markedly contracted. The foot reminded one of a seal's flipper; except for the slight flexion of the toes, it was without muscle control. The legs were of equal length. The atrophy of the left thigh amounted to 2 cm., and the atrophy of the left calf to 2.5 cm. All the muscles were flabby (Figs. 16 and 17).

Treatment: September 10, 1908. Before the patient came to the hospital for treatment, she had been advised to have the foot amputated.

In the writer's opinion, a bone operation was out of the question, because the deformity was of such long standing and the degree of plantar flexion of the toes was so great, that operative interference would endanger the nutrition of the foot. Consequently, the foot was forcibly stretched, and the plantar fascia divided. Correction of the foot to within 15 degrees of a right angle was obtained, making a gain of 130 degrees. In so doing, the skin over the Achilles tendon was ruptured. Plaster was applied from the toes to the groin, with the knee slightly flexed.

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* For technique, see Chapter XIII—Operative Treatment.

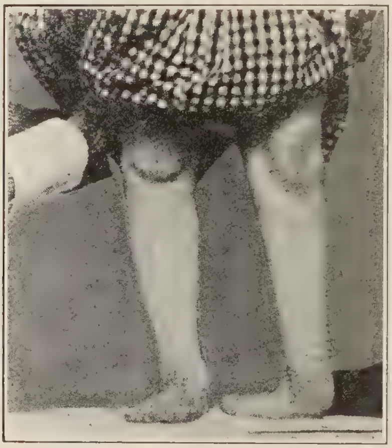

Fig. 16.—Case I. R.R. Talipes equinus. Photograph before operation, showing equinus deformity.

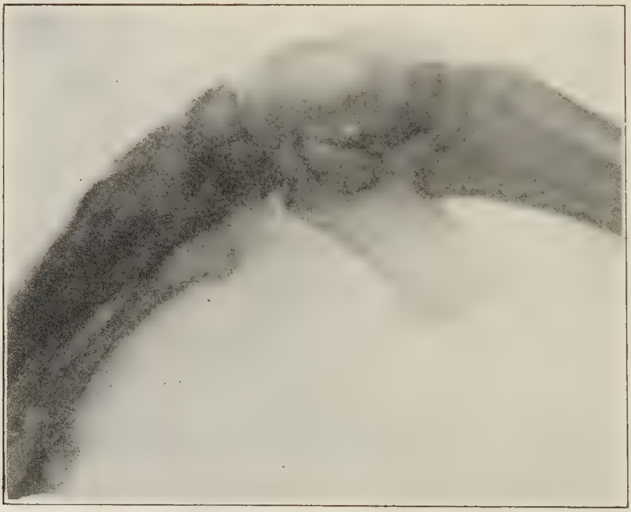

Fig. 17.—Case I. R. R. Talipes equinus. Lateral radiogram before operation.

September 29, 1908. Convalescence uneventful.

November 18, 1908. Another manipulation of the foot was done, in which the remaining 15 degrees of plantar flexion were overcome. Plaster was applied with the foot in normal position.

December 14, 1908. Another manipulation of the foot into dorsal flexion was performed. A plaster bandage was applied with the foot in a slight valgus position and above a right angle.

January 18, 1909. The patient had 25 degrees of motion in the ankle. Some power was demonstrated in the anterior group of muscles. The unexpected return of function was apparently due to the restoration of the foot to normal position. The patient was advised to report three times a week for massage and passive exercises.

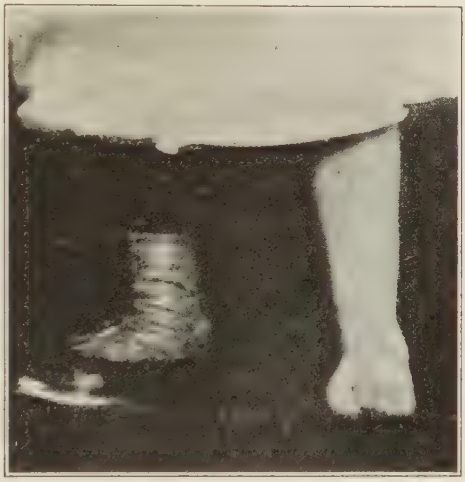

Fig. 18.—Case I. R. R. Talipes equinus. Anterior view after operation.

Result: Perfect function. No deformity (Figs. 18 and 19).

Tenotomy of the Achilles Tendon.—When manipulation fails, tenotomy of the Achilles tendon is a simple procedure by which correction is satisfactorily accomplished in many cases.

Lengthening of the Achilles Tendon.—Adult cases of contracture of the Achilles tendon with extreme deformity are best treated by tendon lengthening. A tenotomy might result in permanent separation of the tendon ends.

Gallie Fixation of the Dorsal Flexors. This operation may be performed in the presence of a strong posterior group of muscles, when for some reason an arthrodesis is not indicated.

Astragalectomy.—In cases in which no power remains in the anterior muscle group, an astraglectomy will increase stability and restore the equilibrium, and at the same time preserve the motion of the foot. This operation is advisable in children from eight to fifteen years of age, as motion is easily preserved during this period. Care must be taken to prevent a recurrent equinus deformity.

Bone Block Operation (Campbell).—In simple equinus deformity resulting from involvement of the anterior group, this block operation may be used in conjunction with stabilization of the mid-tarsal joint. If the mid-tarsal joint is stable, the bone block may be used to correct the toe-drop.

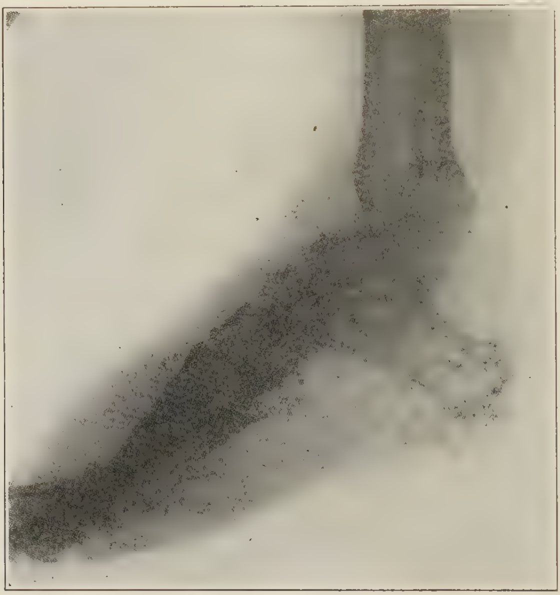

FIG. 19.—Case I. R. R. Talipes equinus. Lateral radiogram after operation.

TALIPES CALCANEUS.

In talipes calcaneus, the foot assumes the position of extreme dorsiflexion. Some varus or valgus deformity usually is found in conjunction with the calcaneus; talipes valgus is the more frequent complication.

Etiology and Pathology.—The posterior group of muscles of the lower leg is paralyzed as the result of damage to the motor cells innervating the posterior tibial nerve. Soon after paralysis occurs, anatomical changes take place in the foot. The anterior muscle group contracts, and in so doing stretches the Achilles tendon and draws the foot into extreme dorsal flexion. The plantar fascia contracts, giving rise to a cavus deformity (Fig. 20). Gradually, the os calcis assumes a vertical position, the heel broadens, and the astragalus becomes arched. The whole foot is displaced forward, and the normal posterior prominence of the heel disappears.

Symptoms.—Plantar flexion at the ankle-joint is weak or entirely lacking, depending upon the extent of the paralysis and the degree of deformity. The patient cannot stand or run on the toes. The limp is often greater than indicated by the degree of paralysis. The gait is awkward, as the normal spring is lost.

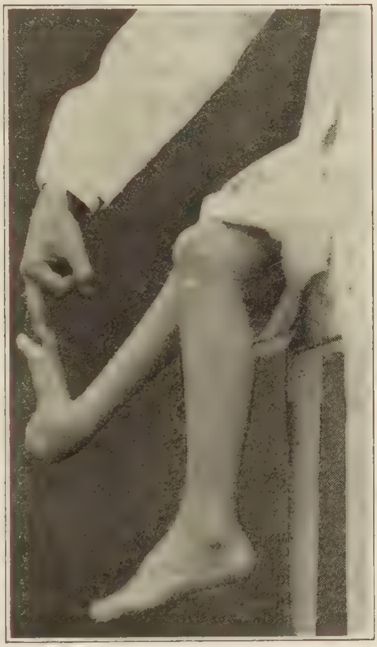

Fig. 20.—Talipes calcaneus.

Diagnosis.—A muscle test reveals the loss of power in the posterior group of muscles. The examiner, holding his finger slightly below the patient's foot, requests him to plantar-flex his ankle. The responsive movement should be carefully observed, to determine whether flexion is due to contraction of the gastrocnemius or to action of the plantar flexors of the toes, which are rarely paralyzed and may produce weak plantar flexion when the patient is not bearing weight.

Prognosis.—The prognosis is good, both as to the correction of deformity and as to the improvement of stability and function. Deformity in its early stage may be overcome by conservative manipulative measures, and in its late stage, by operation. When stability and function are greatly impaired, an astragalectomy or a subastragalar arthrodesis will aid in the restoration of function.

Treatment.—Preventive.—Particular care should be taken to prevent the development of calcaneus deformity, for once it has occurred, correction may be difficult, particularly when secondary contractures of the plantar fascia and changes in the os calcis have taken place. As soon as one has determined a weakness or paralysis of the posterior muscle group, the foot should be placed in the position of extreme equinus, in order to prevent stretching of the weakened muscle group and to favor the return of power. The reason so many cases of talipes calcaneus fail to recover is because the gastrocnemius becomes stretched when the foot is allowed to assume a dorsiflexed position.

A light plaster bandage is the best means of holding the foot in the position of overcorrection. The plaster is split a few days after tenderness has disappeared, to permit local therapy. Baking, hot immersions, massage, and muscle training are beneficial.* In addition, the attendant should manipulate the foot into plantar flexion morning and night, taking care to push the heel posteriorly.

At the end of six weeks the patient may begin to walk on the plaster, the sole of which has been reinforced. The return of power is favored by keeping the foot in the position of plantar flexion for the first six to twelve months, either by means of a plaster bandage or by some form of apparatus.

Conservative Treatment.—The above treatment is used to correct mild calcaneus deformity.

Mechanical†.—In early convalescence, an arthrodesis brace holding the foot in 5 degrees plantar flexion may be worn inside a shoe. Later, when more function is possible, the calcaneus brace or the calcaneus shoe, both of which are equipped with reverse stop-joints to prevent dorsal flexion beyond 90 degrees, will aid function, check progressing deformity, and favor the return of muscle power.

Operative.‡—*Manipulation.*—If deformity continues to develop in spite of conservative measures, it will be necessary to use forcible manipulation to overcome the contracture of the anterior muscle group. Following manipulation, a plaster bandage is applied to hold the foot in the position of plantar flexion. If necessary, the manipulative procedure may be repeated several times.

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* See Chapter XII on the Restoration of Muscle Power.

† For complete description of braces, see Chapter XIV—Apparatus Section.

‡ For complete description of the operative technique, see Chapter XIII on Operative Treatment.

Shortening of the Achilles Tendon.—This operation is used to prevent prolonged stretching of the affected muscle group. The procedure should be used only as a measure of temporary relief, since its results are not permanent.

Transplantation of the Peroneal Tendons into the Achilles Tendon.—In children under eight years of age who show a tendency to develop calcaneus deformity, transplantation of the peronei into the Achilles tendon will aid in checking the displacement of the foot forward and the development of cavus deformity. The value of such an operation is only temporary, and a stabilizing procedure is required later. In children more than eight years old, the transplantation may be used as a supplementary measure to an astragalectomy.

Astragalectomy with Transplantation of the Peroneal Tendons into the Achilles Tendon.—This is the operation of choice in cases of talipes calcaneus between the ages of eight and fifteen years. It ensures stability and increases function. The results are permanent. Transplantation of the peroneal tendons into the Achilles is a valuable supplementary measure.

Subastragalar Arthrodesis.—The choice between this operation and an astragalectomy will depend upon the age of the patient and, to a certain extent, upon the judgment of the surgeon. A subastragalar arthrodesis is the preferable operation in cases between the ages of fifteen and twenty years, and may be advisable in some cases between the ages of twelve and fifteen years.

If there is an element of cavus associated with the calcaneus deformity, it may be corrected by removing a bone wedge from the mid-tarsal joint.

Jones' Operation for Calcaneocavus.—When the foot is so flail that the remaining muscle power is valueless, this operation may be used. It has far better results when performed in children more than eight years of age. If there is a great deal of lateral instability, a subastragalar arthrodesis is done in conjunction with the operation for the correction of the calcaneocavus.

TALIPES EQUINOVALGUS.

Talipes valgus not infrequently is associated with talipes equinus. The equinus deformity develops when the tibialis anticus is paralyzed and the Achilles tendon contracts. The valgus deformity develops when, in the absence of power in the inner muscle group, the peronei, whose action is unopposed, contract (Fig. 21). Paralysis about the knee is often seen in conjunction with equinovalgus deformity.

Symptoms.—Limp and deformity cause considerable disability. Steppage gait is always present, and the patient lifts his foot high to avoid tripping. Atrophy is usually marked. If weight-bearing has been allowed, genu recurvatum may be present. It is difficult to correct the knee deformity.

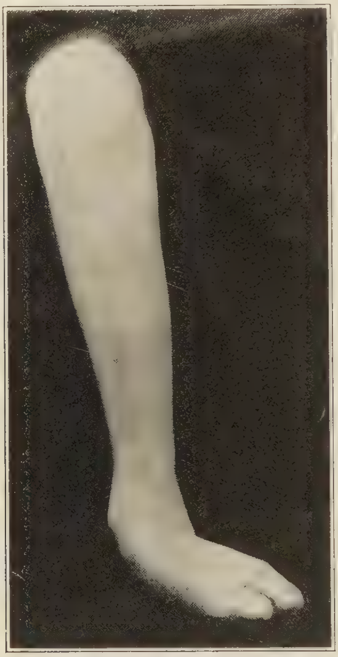

FIG. 21.—Talipes equinovalgus.

Diagnosis.—The muscle tests for dorsiflexion and inversion are used to determine the presence of paralysis of the anterior and inner muscle groups. In old-standing cases, the foot is often in a fixed position, the muscles are atrophied, and there are deformities elsewhere.

Paralytic talipes equinovalgus may sometimes be confused with an equinovalgus of spastic origin. The increased knee-jerks, the spastic gait, and the more rigid type of deformity in the spastic cases serve to differentiate these lesions.

Prognosis.—Provided equinovalgus is treated in the early stages, it may be corrected by conservative measures. Function will improve in many cases after deformity has been corrected. Extreme or neglected deformities may be corrected by operative measures. When instability persists, stabilizing procedures are very successful in reëstablishing function.

Treatment.—PREVENTIVE AND CONSERVATIVE.—If the distribution of the paralysis is studied carefully early in the attack, it is always possible to prevent the development of deformity by maintaining the foot in overcorrection in a plaster bandage. As soon as tenderness disappears, the plaster is bivalved, and local therapy begun. In the course of four to six weeks, weight-bearing on the plaster should be encouraged.

These conservative methods may also be used to correct mild deformity.

MECHANICAL*.—If any trace of weakness or deformity remains when the plaster is removed, it is advisable to use some form of mechanical apparatus for support. In the more severe case, an equinovalgus brace is worn day and night to hold the foot in the position of varus and to prevent contraction of the Achilles tendon. In the majority of cases, an equinovalgus shoe is substituted during the day, and the brace worn only at night. This shoe, combined with a Whitman brace or a raised heel and sole, holds the foot at a right angle and in the position of slight varus.

It is important that the patient should continue to wear support until complete recovery has taken place, or until operative interference is indicated.

OPERATIVE.†—Operative interference is required for the correction of deformity when conservative treatment is ineffective, and it is necessary in cases in which deformity has been held in check, but in which regeneration has not occurred. If possible, stabilizing procedures should be postponed until two years have elapsed from the onset of the paralysis, and until the child is eight to ten years of age.

Manipulation.—Manipulation may be used to correct equinovalgus deformity in its early stage. It may also be used in old-standing cases as a preliminary measure to surgical treatment.

Tenotomy of the Achilles Tendon. When it is impossible to overcome a contraction of the Achilles tendon by manipulation, a tenotomy may be done. This operation may be performed at any stage of convalescence.

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* For details, see Chapter XIV—Apparatus Section.

† See Chapter XIII on Operative Treatment for details.

Tendon Lengthening.—In adult cases of extreme equinus deformity, elongation of the Achilles tendon may be advisable.

Gallie Fixation Operation.—In equinovalgus deformity, tenodesis is of value when used in conjunction with an astragalo-scaphoid arthrodesis.

Wedge Osteotomy.—If the deformity is of long-standing, it may be necessary to remove a wedge from the astragalo-scaphoid region to secure correction of the valgus deformity.

Astragalo-scaphoid Arthrodesis.—This procedure is indicated when the valgus deformity continues to progress in spite of conservative treatment. It may be performed before the two-year period has elapsed, and before the child reaches the age of twelve years. Its advantage lies in holding the deformity in check until further stabilizing operations are indicated. In some instances it may be advisable to supplement the arthrodesis by transplantation of the peroneus longus into the scaphoid.

Loop Operation (Whitman).—When the outer dorsiflexors are in good condition, this operation may be used.

Astragalectomy.—In cases in which deformity has been corrected, but in which instability persists because of poor musculature, astragalectomy aids in restoring the equilibrium. It is particularly applicable to cases from eight to fifteen years of age, since during this period ample motion is preserved.

Subastragalar Arthrodesis.—When there is general involvement of all the foot muscles in addition to extreme paralysis of the inner group, a subastragalar arthrodesis is the operation of choice. It is most frequently used in cases from twelve to twenty years of age. All deformity should be corrected before this operation is performed.

Bone Block Operation (Campbell).—Campbell recommends the use of his bone block operation in equinovalgus deformities. Tarsal resection, which is done to correct the valgus, provides bone for the blocking.

TALIPES EQUINOVARUS.

Talipes equinovarus develops when the peroneal muscles as well as the dorsiflexors are weakened or paralyzed (Fig. 22). The involvement of the anterior leg muscles is the result of damage to the anterior tibial nerve, and the paralysis of the peroneal muscle group is due to damage to the musculocutaneous nerve. The first step in the development of equinovarus deformity is the sagging of the forefoot. Then cavus develops, its severity depending upon the extent of the paralysis and the degree of contraction of the

Achilles tendon and plantar fascia. In the absence of power in the peroneal group, the tibialis positicus and the anterior tibial muscle group, if it has any power, exert an unopposed pull and give rise to the development of a varus deformity.

In time, bone changes take place, occurring especially at the mid-tarsal joint. A large bursa is found under the cuboid in extreme cases. If the deformity is of long-standing, it is very difficult to secure correction by conservative means.

The toes show contractures; in some cases all the toes are in plantar contraction, and in other cases only the great toe is involved. The leg shows marked atrophy as the result of disuse. In deformities of long-standing, shortening and circulatory changes of the lower leg are not uncommon. Ulcers may develop in neglected cases.

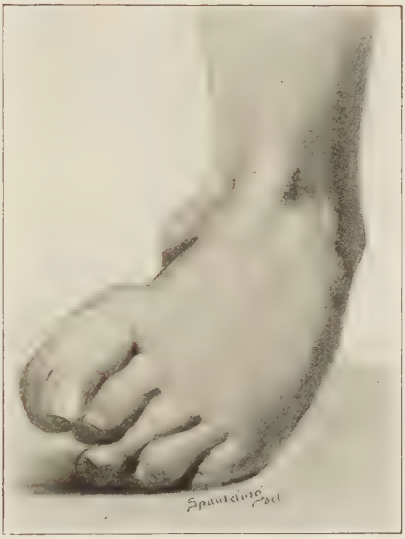

Fig. 22.—Talipes equinovarus.

Symptoms.—Limp and deformity cause considerable disability. The leg apparently is lengthened by the talipes equinus, so that in walking the patient lifts the knee high to avoid stumbling. In addition, the foot rolls out in standing.

Diagnosis.—The absence of the dorsiflexors and peronei may be determined by the muscle tests for dorsiflexion and eversion.

Paralytic equinovarus may be confused with congenital club-foot. In the latter deformity, the varus is generally more pronounced than the equinus; in cases of paralytic origin, the reverse is true. In congenital club-foot, the deformity involves the entire tarsus, whereas in paralytic equinovarus the condition is due to the subluxation of the joints. One of the signs indicative of a paralytic equinovarus is the irregular arrangement of the toes; in congenital club-foot the toes are usually in normal relation. In paralytic cases there is likely to be involvement elsewhere. The atrophy of the calf and shortening of the leg are more pronounced than in congenital varus deformity. The history of the case also enables one to differentiate between the two types.

Prognosis.—If treated early, the deformity can be controlled by conservative or simple operative measures. After correction of the malposition the return of power may approach normal. When stability is greatly impaired, transplantation of the anterior tibial tendon into the cuboid, or astragalectomy with fixation of the distal peroneal tendons to the fibula, ensures stability and increases function.

Treatment.—PREVENTIVE AND CONSERVATIVE.—As soon as any weakness of the peroneal and anterior muscle groups is evident, steps should be taken to maintain the foot in an overcorrected position, in order to prevent the development of deformity and to favor the return of muscle power. A light, well-padded plaster bandage is the most efficient means of maintaining the foot in calcaneovalgus. The plaster bandage is worn until recovery has taken place, or until the use of apparatus is indicated; the duration of the period varies from six weeks to six months. Weight-bearing on the plaster bandage is permitted during this period, the amount depending upon the extent of the paralysis, the rate of recovery, and the general condition of the patient.

As a general rule, the plaster bandage is split in ten days to allow local therapy. If deformity tends to recur, it is well to maintain overcorrection for several weeks, without bivalving the plaster.

Manipulation is a satisfactory procedure in overcoming early contractures. In the absence of the anterior and peroneal muscle groups, the attendant should push the foot gently into calcaneovalgus fifty times each morning and night. During the process, care should be taken not to allow the foot to revert to a position of deformity. Deformity may be controlled by an attendant who conscientiously carries out these manipulations.

MECHANICAL*.—When the plaster bandage is discarded, some form of light apparatus is used to prevent muscle-tire in limbs that are regaining power, or to hold the foot in an overcorrected position until regeneration takes place, or until operative treatment is advisable.

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* For details, see Chapter XIV—Apparatus Section.

In early convalescence, the equinovarus brace, which holds the foot in valgus and prevents it from dropping below a right angle, may be worn day and night. In mild cases requiring only moderate correction, or in cases which have recovered sufficiently, the equinovarus shoe may be worn in the daytime in place of the cumbersome brace. The shoe is lighter than the brace and easier to apply. The patient should continue to wear the brace at night until muscle balance has returned.

OPERATIVE.*—Surgical treatment is indicated when deformity recurs in spite of conservative treatment, or when function is imperfect at the end of two years from the onset of the attack.

Forcible Manipulation.—When conservative measures are not sufficient to secure overcorrection in the early stages, the foot may be forcibly manipulated into calcaneovalgus and retained in this position by a plaster bandage. Manipulation may also be used to overcorrect any slight deformity before operative interference is undertaken.

Tenotomy of the Achilles Tendon.—Occasionally, when manipulation and stretching of the Achilles tendon are not sufficient to correct equinus deformity, it is advisable to tenotomize the Achilles tendon. The results of this procedure are most satisfactory; non-union is rare, occurring only in adult cases when the tendon sheath has been divided and the foot held in too great overcorrection. It is very important to overcorrect the varus deformity before performing a tenotomy; otherwise, the leverage so necessary in correction of varus deformity is lost, and calcaneovarus develops.

Tendon Elongation.—This procedure may be used in adult cases when contraction of the Achilles tendon is so extreme that it would be difficult to obtain full correction by tenotomy. Such an operation is required in very few cases.

Transplantation of the Anterior Tibial Tendon into the Cuboid.—This is practically the only satisfactory tendon transplantation performed for paralysis of the foot. It is used in cases of weakness or paralysis of the peroneal muscle group, in which deformity has been corrected, but in which there still exists a tendency for the foot to return to the position of equinovarus.

Gallie Fixation.—Fixation of the peroneal and dorsal flexor tendons occasionally is advisable in cases of general muscle weakness, when a tendon transplantation would not be sufficient to restore stability, and when a stabilizing operation is not indicated.

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* For details of operative technique, see Chapter XIII on Operative Treatment.

Wedge Out of the Astragalus.—If actual structural deformity has taken place in the astragalus, a change that is likely to occur in neglected cases, excision of a wedge-shaped piece of bone from the astragalus may be necessary to overcorrect the deformity. This procedure is sufficient to restore the foot to good position. It is usually performed as a forerunner to a stabilizing procedure.

Astragalectomy with Fixation of the Peroneal Tendons into the Fibula.—The results of this operation are most satisfactory. Not only is stability increased, but the fixation of the peroneal tendons provides against a tendency to the recurrence of varus deformity. The operation is of particular value in cases of instability resulting from abnormal lateral movement in the astragalo-tibial joint, or from general weakness or paralysis of all muscle groups of the foot.

Subastragalar Arthrodesis.—When the condition is such that an astragalectomy is not advisable, or when the age of the patient contraindicates its use, a subastragalar arthrodesis may be performed to restore stability. Much of the lateral instability is overcome by this procedure, and the equinus deformity is somewhat compensated.

Bone Block Operation for Drop-foot (Campbell).—This check operation may be used when the child is over eight years of age. The varus deformity is corrected by tarsectomy, and the bone piece removed is used to form the posterior block to control the equinus deformity.

TALIPES CALCANEOVALGUS.

Calcaneus deformity usually is accompanied by some degree of valgus; the resulting combination is one of the most disabling deformities found in paralytic foot conditions (Fig. 23). Fortunately, the treatment of such deformities is most satisfactory.

Etiology and Pathology.—Calcaneovalgus deformity develops as the result of paralysis of the inner and posterior muscle groups, which become involved when the posterior and anterior tibial nerves are damaged. Valgus deformity usually develops first, the degree depending upon the extent of the paralysis. Soon after, the foot becomes fixed in dorsiflexion, due to the contraction of the dorsal flexors. Weight-bearing tends to increase the deformity. The heel broadens, and the weight-bearing line of the tibia and fibula is transferred backward.

Symptoms.—Limp and deformity are marked and greatly limit activity. Atrophy of the limb may be considerable. If the case is seen late in convalescence, genu recurvatum may be present as a secondary deformity.

Diagnosis.—The diagnosis is made from the history and the clinical symptoms of extreme limp, unstable gait, deformity, atrophy, and loss of power to invert and plantar-flex the ankle.

Prognosis.—In the early stages, the deformity may be corrected, and function restored to a certain extent by conservative measures. In the late stages, when the deformity is structural, excellent functional results are obtained by operative treatment.

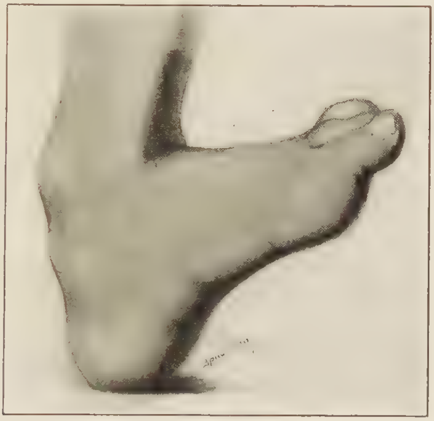

FIG. 23.—Talipes calcaneovalgus.

Treatment.—CONSERVATIVE.—Early treatment consists of maintaining the foot in a plaster bandage in the position of equinovarus. After a period of about ten days to three weeks, the plaster bandage is bivalved to permit local treatment. Baking, immersions in hot water, and massage are advisable as soon as the tenderness disappears. The attendant should also manipulate the foot into equinovarus fifty times each morning and night, to prevent contractures. As soon as the acute stage has passed, the patient should be encouraged to walk on the plaster, to which a felt heel has been attached.

MECHANICAL*.—After the removal of the plaster, a calcaneovalgus brace is worn day and night. This support maintains the foot in varus and prevents motion above a right angle. A raised heel and sole used in conjunction with the brace assists in combatting the valgus element.

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* For details of braces, see Chapter XIV—Apparatus Section.

When more activity is permissible, a calcaneovalgus shoe may be worn for walking purposes. The child should continue to wear a light brace at night to maintain the foot in the position of overcorrection.

Operative*.—Surgical measures are indicated when conservative treatment has failed to prevent the progression of deformity, or when deformity has been corrected, but the muscle balance is still unstable. The end-results of operative treatment in calcaneovalgus are excellent, and the patient is assured of an increase in function and stability, as well as relief from deformity.

As in other deformities, the surgical procedure selected for a given case of calcaneovalgus depends upon the age of the patient and the judgment of the surgeon.

Manipulation.—Forcible manipulation of the foot into equinovarus, followed by the application of a plaster bandage, is often effective in overcoming early contracture deformities. The procedure may be repeated several times before open operation is attempted.

Transplantation of the Peronei into the Achilles Tendon.—This operation, when used in early cases of calcaneus, will often check the deformity temporarily. In cases of long-standing, the transplantation when performed alone is of little value, since it does not overcome the forward displacement of the foot; when combined with a stabilizing operation, the transplantation is of value in establishing greater stability. This transplantation is a most successful adjunct to an astragalectomy.

Fixation of the Achilles Tendon and the Tibialis Posticus (Gallie).—This surgical measure may be used in young children to hold the foot in position temporarily. It is seldom necessary to perform the operation, however, as apparatus will usually check deformity until the child reaches the age when an operation that ensures permanent results may be performed.

Astragalo-scaphoid Arthrodesis.—When conservative measures fail to check valgus deformity, an astragalo-scaphoid arthrodesis is of value. Either a transplantation of the peronei into the scaphoid, or a fixation of the anterior tibial tendon into the tibia, may be combined advantageously with this arthrodesis.

In young patients, these surgical procedures may afford only temporary relief, and the deformity is likely to recur within a few years. They have the advantage, however, of checking the progression of the deformity until more extensive operative procedures are advisable.

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* For details of operative technique, see Chapter XIII.

A Whitman brace and raised heel and sole should be worn to prevent strain after an astragalo-scaphoid arthrodesis.

Astragalectomy with Transplantation of the Peronei into the Achilles Tendon.—Probably no operative procedure devised for paralytic conditions is followed by more strikingly satisfactory results than astragalectomy with transplantation of the peronei into the Achilles tendon. In cases from eight to fifteen years of age, it results in excellent stability and has the advantage of preserving motion. The value of an astragalectomy, when properly done, cannot be overestimated.

Subastragalar Arthrodesis.—This operation is particularly applicable in cases from fourteen to twenty years of age, in which astragalectomy would not be advisable because of the possibility of the persistence of tenderness. Stability and function are increased materially by performing a transplantation of the peroneal tendons into the Achilles tendon in combination with the subastragalar arthrodesis.

TALIPES CALCANEOVARUS.

A varus deformity is seen occasionally in association with talipes calcaneus, and the combination is a very disabling deformity (Fig. 24). If such cases are treated promptly in the early stages, correction may be accomplished, but old-standing calcaneovarus deformity is very difficult to overcome.

Etiology and Pathology.—The deformity occurs when both the outer and posterior muscle groups are paralyzed. The condition develops in much the same manner as simple paralytic varus and calcaneus. When the peronei are weakened, the muscles on the inner side of the foot, being unopposed, undergo contraction and pull the foot into varus. In the absence of the posterior muscle group, the anterior leg muscles contract, the Achilles tendon is stretched, and the foot assumes a dorsiflexed position.

If no attempt is made to correct the malposition of the foot, the deformity eventually becomes structural. The neck of the astragalus twists, the scaphoid and the entire tarsus rotate inward, and the os calcis assumes a vertical position. The heel is broadened, and its posterior prominence is lost.

Symptoms.—The limp is greatly exaggerated by instability. The patient walks on his heel. Deformity, especially the calcaneus element, tends to increase rapidly. The calf is markedly atrophied, due to paralysis of the posterior group. The toes are contracted when power remains in the plantar flexors. Hallux rigidus is common.

Diagnosis.—The loss of muscle power in the posterior and outer groups may be determined by muscle tests. The patient walks on the heel with a marked limp.

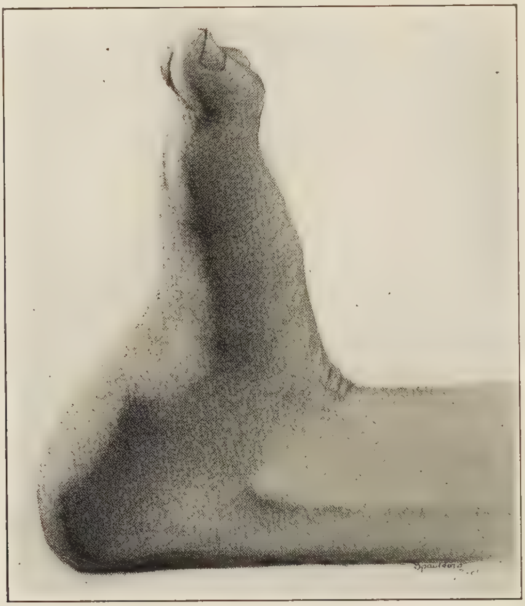

FIG. 24.—Talipes calcaneovarus.

Prognosis.—If the deformity is recognized very early it may be corrected by conservative methods. Old-standing cases are difficult to correct, and the results are not promising. Stabilizing operations are of value in helping to increase function in unstable feet, provided varus deformity has been corrected.

Treatment.—PREVENTIVE AND CONSERVATIVE.—Deformity may be prevented if the case is treated carefully from the beginning of the attack. In neglected cases in which deformity has developed, further progression may be avoided by proper care. Conservative treatment consists of the application of a plaster bandage with the foot held in the position of equinovalgus. The plaster remains on during the early convalescent stage, except when it is removed for local therapy.

Baking, hot fomentations, and massage are begun as soon as the tenderness disappears. Gentle manipulation into equinovalgus should be started early. As soon as improvement takes place, passive motions and muscle training are of value in teaching the child to use his muscles more accurately and intelligently.*

MECHANICAL†.—In the course of six weeks, the plaster bandage is removed, and a calcaneovarus brace substituted. It is worn day and night, being removed only during local treatments.

As soon as more active use of the foot is advisable, a calcaneovarus shoe is worn during the day, and the brace applied only at night. A raised heel and sole on the outer side of the shoe increases its efficiency.

OPERATIVE‡.—It becomes necessary to operate when conservative measures are not preventing deformity, or when, after a period of two years, the muscle power has not returned.

The type of operation indicated in a given case varies according to the mechanical problem presented by the foot.

Manipulation.—Forcible manipulation often lessens mild contractures. The foot should be manipulated into equinovalgus and maintained in this position for several months by a plaster bandage. Manipulations should be repeated until all varus deformity has been overcorrected. In long-standing cases, it may be very difficult to secure overcorrection by manipulation.

Tendon Shortening.—Occasionally, the shortening of the Achilles tendon may be considered to prevent the posterior muscles from becoming permanently stretched.

Wedge Osteotomy. Severe varus deformity may be corrected by the removal of a wedge-shaped piece of bone from the outer side of the tarsus.

Astragalectomy.—An astragalectomy supplemented by fixation of the peroneal tendons into the fibula is a satisfactory stabilizing measure. It is well to correct the varus deformity, and to hold the foot overcorrected for a few months, before the astragalectomy is done. Care should be taken to maintain the foot in the position of valgus following astragalectomy. If such precautions are taken, there is no danger of the recurrence of varus. The failure of this stabilizing measure in calcaneovarus deformity is due to insufficient correction of the varus before astragalectomy and to improper after-care.

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* See Chapter XII on the Restoration of Muscle Power.

† For details, see Chapter XIV—Apparatus Section.

‡ For details, see Chapter XIII on Operative Treatment.

Subastragalar Arthrodesis.—In long-standing cases in which cavus has developed, and particularly in adults, a subastragalar arthrodesis is the operation of choice. Correction of cavus or varus may be accomplished at the time of arthrodesis, by removing a plantar wedge.

FLAIL-FOOT.

Flail-foot, or dangle-foot, is the result of complete or nearly complete paralysis of all the muscles of the lower leg. Total paralysis is rare, and slight power usually remains in one or more muscle groups.

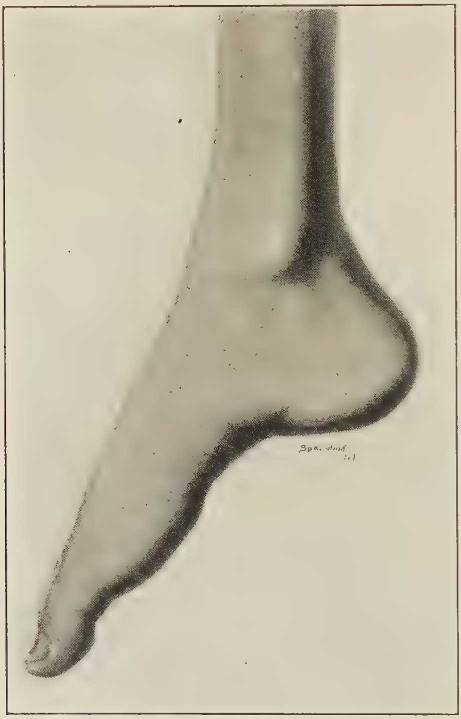

FIG. 25.—Flail-foot, showing toe-drop.

Some element of deformity is always found in a flail-foot, due to the influences of gravity, the contraction of the soft parts, improper habit postures, and the pull of the muscles in which any power remains. In extensive involvement, the foot drops under the force of gravity, and an equinus deformity develops (Fig. 25). If slight power remains in the plantar flexors, this action plus the force of gravity produces toe-drop, which is accompanied usually by contraction of the toes and by a slight degree of cavus, or in rare cases, by slight varus deformity (Figs. 26 and 27). The presence of power in the anterior muscle group, if unopposed, favors the development of talipes calcaneus.

The contraction of the soft parts is a potent factor in the development of deformity. As soon as the plantar fascia contracts, cavus develops; if it becomes extreme, correction is often difficult.

Weight-bearing, without proper protection of the extremity, allows the foot to assume positions of deformity, or aggravates malpositions that have already begun to develop. Varus deformity predominates when the patient is ambulatory, since even the slightest remaining power in the plantar flexors tends to invert the foot. Structural changes occur rapidly under the influences of weight-bearing. Walking with the foot in a position of equinus produces changes in the astragalus; it becomes shortened, assumes a camel-back shape, or is displaced forward so that the heel rides nearly under the tibia. The foot becomes shortened, and the forefoot and heel broadened. Not uncommonly, knee-joint deformities, such as knock-knee or recurvatum may be found associated with flail-foot.

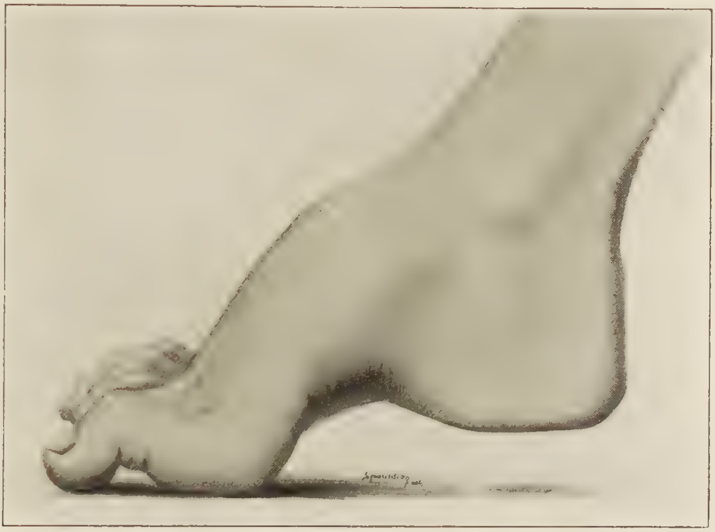

Fig. 26.—Flail-foot, showing contraction of the Achilles tendon and contraction of the toes.

Symptoms.—The disability from flail-foot is often excessive. When walking is possible, it is labored, particularly if deformity is present. If other parts of the same leg or of the opposite leg are paralyzed, the patient will experience great difficulty in getting about. When the flail-foot is associated with thigh paralysis, the patient is greatly incapacitated, and weight-bearing may not be possible. Marked atrophy is often present.

Diagnosis.—A diagnosis of flail-foot is made from the extreme limp, loss of muscle power, and atrophy, following an attack of poliomyelitis.

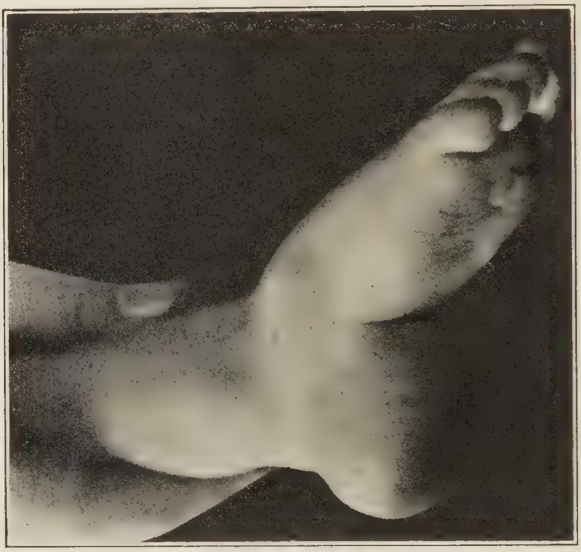

Fig. 27.—Flail-foot, showing cavus deformity.

Prognosis.—The prognosis of flail-foot depends upon the establishment of stability. The foot may be successfully stabilized by several methods.

Treatment.—Preventive and Conservative.—The treatment of dangle-foot is concerned not only with the flail condition of the foot muscles, but also with the prevention and correction of contractures and deformity.

Upon the onset of paralysis, care should be taken to prevent the development of any deformity by protecting the foot from the weight of the bed-clothes. As soon as tenderness permits, the leg is placed in a light plaster bandage with the foot at a right angle. Within a few days, the plaster is bivalved to permit local therapy.*

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* For details of local treatment, see Chapter XII on Restoration of Muscle Power.

If no deformity develops, walking on the plaster bandage may be started about six weeks after the onset of paralysis.

MECHANICAL*.—In time, some form of apparatus supersedes the plaster bandage. The arthrodesis brace, which may be worn inside a shoe, is most efficient in preventing motion at the ankle-joint. When the condition warrants, a shoe with an outside upright allowing limited motion may be used. Walking is less awkward with a brace that allows some motion.

OPERATIVE†.—Many cases of flail-foot come to operative interference, as stabilization is imperative, and deformities, which develop during the convalescent period, require correction. Mild deformities may be overcome by repeated manipulative procedures, followed by the application of plaster bandages to hold the foot in an overcorrected position. Contracture of the plantar fascia may be overcome by a fasciotomy and stretching. Fixed deformity may require a wedge osteotomy for correction.

Once the malposition has been corrected, the problem is to stabilize the foot so that it will be maintained permanently in normal position. Stabilization is likewise necessary in an unstable foot in which no deformity has developed. The selection of the operation depends upon the extent of the muscle involvement, the age of the patient, and the surgeon's experience. If no deformity has occurred, a stabilizing operation should be withheld until two years have elapsed from the onset of paralysis, and until the child is eight to twelve years of age. If an arthrodesis of the ankle-joint is required, it should not be performed until the patient is sixteen years old.

Astragalectomy.—In children between eight and fifteen years of age, astragalectomy gives very satisfactory results. It not only stabilizes the foot, but also preserves motion.

Subastragalar Arthrodesis.—The functional results of this operation are most gratifying, particularly if there is any power remaining in the anterior muscle group. This is the operation of choice in patients between fifteen and twenty years of age, and may be used in some cases between the ages of twelve and fifteen years, when there is a general muscle weakness with only slight instability of the ankle.

Arthrodesis of the Ankle-joint.—In older patients, arthrodesis of the ankle-joint is preferable to an astragalectomy or a subastragalar arthrodesis. Objection may be made to stiffening a joint, but the patient can walk much better with a stiff joint when the foot is in proper position, than when the foot is unstable, or burdened with cumbersome apparatus. After an arthrodesis, the function is greatly increased; many patients do not limp at all, and others only slightly.

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* For details, see Chapter XIV—Apparatus Section.

† For description of the operative technique, see Chapter XIII.

PARALYSIS AFFECTING THE KNEE.

Paralysis in the region of the knee is of very common occurrence. It is more frequently partial than total; even in extensive destruction, it is very seldom that some fibers of the flexors, extensors, or adductors do not escape. Lovett,[1] in a study of the relative involvement of all muscles, noted that affection of the quadriceps led in frequency. Extensive paralysis in the hip or the foot is usually found in connection with weakness of the thigh muscles.

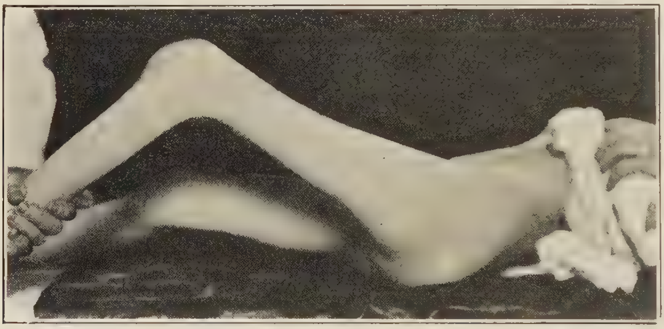

FIG. 28.—Contracted hamstrings.

The disability resulting from paralysis about the knee may be exceedingly great. The leg is very unstable, and the patient is constantly fearing that it will give way, causing him to fall. To ensure greater stability in walking, he throws the lower leg forward, and by placing his hand on his thigh exerts pressure to hyperextend the leg and hold the knee locked. In order to place his hand on the thigh, he must bend over considerably, with the result that hip contractures and spinal curvatures frequently develop.

The same effect from unopposed muscle pull is found in paralysis about the knee, as is present in paralytic conditions elsewhere—namely, in the absence of power in one muscle group, its antagonist exerts a pull that is not counteracted, and deformity results. Flexion of the knee occurs when contraction of the hamstrings takes place in the presence of paralysis of the anterior muscle group (Fig. 28). Genu valgum (Fig. 29), another deformity of the knee, develops in old-standing flexion deformity. A third deformity is recurvatum, which develops when both the quadriceps and posterior muscle groups are affected. A fourth deformity is eversion of the lower leg, which occurs most frequently in quadriceps paralysis associated with weakness of the inner hamstring. The presence of deformity in the knee seriously affects the trunk statics, with the result that postural changes occur about the hip and in the spine (Fig. 29). Eventually these changes become structural.

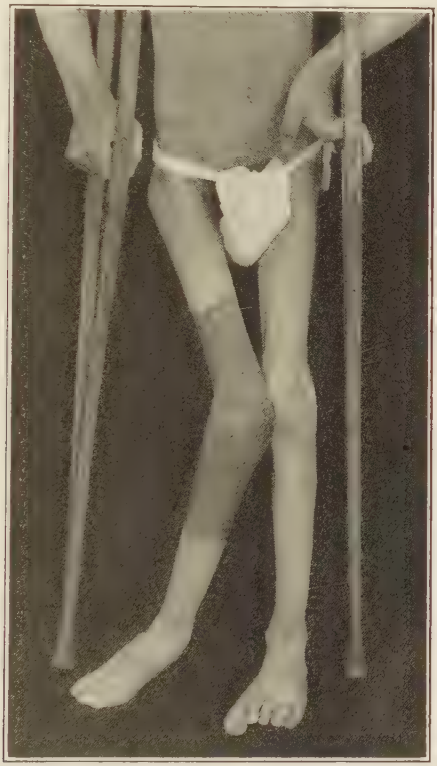

FIG. 29.—Genu valgum and contraction of the iliopsoas.

FLEXION DEFORMITY.

Etiology.—In acute cases of extensive involvement, contraction of the knee may occur early in convalescence, due primarily to the attitude that the child assumes to secure relief from pain. When flexion deformity of the knee occurs late in convalescence, it is the result of the contraction of the active posterior muscles. If the deformity is not overcome in its early stages, the knee may become permanently contracted, and the flexion may be so extreme that the patient crawls on his knees. The deformity is often aggravated by the presence of foot deformities, especially that of talipes valgus.

If the knee is allowed to remain in this flexed position, secondary deformities develop. It is not unusual to see contraction of the iliopsoas associated with flexion of the knee (Fig. 29). In long-standing flexion deformity, genu valgum and subluxation of the tibia develop as secondary deformities. The presence of these sequelæ is a serious complication to the restoration of the function of the limb.

Prognosis.—Flexion deformity interferes with locomotion, and is a particularly serious handicap when there is paralysis elsewhere. If the contracture is treated in its early stages, it may be satisfactorily overcome by conservative and mechanical measures. In cases of severe contraction, or in cases of long-standing, operative measures are necessary to correct the deformity.

In unstable cases, apparatus is of advantage in stabilizing the knee. In properly selected cases, tendon transplantations assist in reëstablishing stability and function.

Treatment.—Preventive and Conservative.—Flexion deformity of the knee should be anticipated, and its development prevented. Early recognition of group paralysis about the knee-joint is most important from the point of view of treatment.

One of the first measures taken to prevent deformity is the application of a plaster bandage extending from the ankle to the hip, with the knee-joint at 180 degrees. The bandage should be worn for a period of ten days to three weeks. It is then split, and if muscle tenderness has disappeared, local treatment of the thigh and leg is started. Any tendency to knee contraction during the early stages may be overcome by careful manipulation. The mother may learn to carry out this simple procedure.

When flexion deformity develops from lack of preventive measures, the contracture may be corrected by careful conservative treatment. This comprises gentle manipulations and the use of traction, followed by massage, exercise, and other stimulative treatment, which improves the circulation and tones up the general musculature. Deformity of practically any degree can be corrected by this conservative treatment, and it is only in cases of extremely resistant deformity that operative interference is required.

Plaster wedging is another means of correcting flexion deformity. A plaster bandage is applied to the leg, from the toes to above the pelvis. When dry, the bandage is cut transversely back of the knee-joint, and a hinge of plaster is left anteriorly. The opening is progressively widened by inserting wedges. Recent cases may be corrected within a week, and moderate cases within a month. Extreme cases require a much longer period for correction.

MECHANICAL*.—Apparatus may be used in paralysis of the knee joint for several purposes: It helps to prevent deformity; it assists in the correction of mild contractures or deformities; and it is of great advantage in stabilizing the knee to aid locomotion. In some instances, the application of a knee brace permits a patient, who has practically total loss of power in the leg, to walk with the aid of a cane or crutches.

When weight-bearing is not a problem, a simple form of caliper or a leather leglet may be used. The caliper is equipped with a knee-cap that helps to correct moderate knee contracture. The leglet is more efficient than the caliper; also it may be equipped with a joint to allow motion. Unfortunately, the expense of making the leglet is so great that it is not always possible to advise it. If only very slight support at the knee is required, a Jones knee brace may be used.

When weight-bearing is a problem, a Thomas ring caliper is the most efficient form of support. A drop-catch, which can be unlocked, may be added to this brace to allow flexion of the knee in the sitting position. In knee paralysis complicated by foot deformity, the simple caliper and Thomas ring caliper brace may be used in combination with Thomas heels, raised heels and soles, and T-straps. Straps about the pelvis are used to control inversion or eversion of the leg.

OPERATIVE†.—The use of operative measures in paralytic conditions about the knee should be advised only after careful consideration. The success of the surgical procedures depends upon perfect asepsis, careful technique, and proper after-care.

Operations are indicated at any stage to correct deformity, when it is evident that conservative measures have not overcome the malposition. Transplantations help to increase stability. Putti[2] recommends a tibio-astragaloid arthroresis to stabilize the knee, when extension is weak or absent as the result of quadriceps paralysis.

Forcible Manipulation.—If deformity is progressing in spite of conservative measures, stretching of the posterior muscle group under ether is indicated at any stage of treatment. Forcible manipulation may also be used as a preliminary step in a tendon transplantation. Care should be taken to avoid extreme stretching, which might result in injury to the popliteal nerves.

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* For details of braces, see Chapter XIV.

† For details of operative technique, see Chapter XIII.

Division of the Hamstring Tendons.—In very severe contractures, which cannot be overcome by manipulation, division of the hamstring tendons is advisable. The tenotomy should be performed by the open method.

Osteotomy for Genu Valgum.—If genu valgum cannot be controlled by means of apparatus, an external osteotomy is advisable. The results are very satisfactory.

Tendon Transplantations.—When deformity has been corrected, but weakness of the muscle groups persists, tendon transplantations may be done to increase the stability of the leg. The value of such procedures should not be overestimated, for as a general rule they do not increase the actual power of extension of the leg to any degree. Their advantage lies in increasing stability, so that a patient no longer feels the necessity of pressing his hand on his thigh when walking. In the rare cases in which sufficient power remains in the quadriceps, transplantations may aid extension, even to the degree of making it possible for the patient to extend his leg from the flexed position.

The sartorius, the hamstrings, or the tensor vaginæ femoris may be used in transplantation. The sartorius lends itself best to transference, and fortunately it frequently escapes paralysis. When the sartorius is so weak that it cannot be used in transplantation, one or both hamstrings may be transferred. Occasionally, a case is seen that warrants the transplantation of the tensor vaginæ femoris.

Putti's Tibio-astragaloid Arthroresis.—This operation aids in stabilizing the knee, by limiting dorsal flexion of the foot by means of a bony block built up at the front of the ankle-joint.

GENU VALGUM.

Several factors enter into the *etiology* of genu valgum. The most important is long-standing flexion deformity, which is often seen as the result of paralysis of the quadriceps. When the leg is in a flexed position, there is more lateral sway than when the limb is straight, and the pull of the strong outer muscle group, in particular that of the tensor vaginæ femoris, influences the development of knock-knee deformity. Genu valgum is also likely to develop in a flail knee, when slight power remains in the tensor vaginæ femoris or in the outer hamstring. If weight-bearing is permitted when flexion and eversion are present, there is a strong tendency for the deformities to increase. Genu valgum is aggravated by deformities of the foot.

The *prognosis* as to the correction of deformity is good. The early cases may be controlled by apparatus, and the older cases corrected by operation. Once deformity has been corrected, function improves.

Treatment.—Anticipation of the development of flexion deformity, and the application of a plaster bandage to prevent its occurrence would naturally avoid the development of genu valgum. If the deformity develops from neglect of treatment, mild cases may be overcome by manipulation and traction, and recurrence prevented by the application of a caliper brace supplied with a knock-knee strap by which leverage is secured. A knock-knee brace accomplishes the same purposes.* Repeated manipulations under ether may be necessary to effect correction in some cases. If knock-knee is greater than 5 to 8 degrees, one must resort to an external osteotomy, a procedure that gives satisfactory results.

GENU RECURVATUM.

Genu recurvatum develops when paralysis of the quadriceps is associated with a weak or paralyzed posterior muscle group. It usually occurs in extensive cases of paralysis in which all the joint structures have become lax. The presence of a talipes equinus tends to aggravate this deformity.

The patient with a paralyzed quadriceps, fearing that the knee will give way, attempts to make the leg stable by locking the knee-joint in hyperextension before bearing weight. When the leg is hyperextended, the posterior capsule is loosened, and the weak posterior muscles are stretched.

The prognosis is not good. Apparatus must be worn constantly to prevent the hyperextension from increasing. Slight deformity may be corrected by wearing apparatus, but the severe case requires permanent use of a brace.

A caliper with a posterior knee strap or a Jones knee brace is a satisfactory form of support. Either splint may be equipped with a lock-joint to prevent hyperextension.†

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* For details of the braces, see Chapter XIV.

† For details, see Chapter XIV.

EVERSION OF THE LOWER LEG.

An outer twist takes place in the tibia in many old-standing cases of paralysis of the quadriceps, when the patient has developed the habit of holding his leg externally rotated in walking. The eversion is more likely to develop if there is any subluxation or flexion deformity present.

An osteotomy may be necessary to correct the rotation deformity.

FLAIL-KNEE.

A dangle-knee is due to severe paralysis of the muscles about the knee. In spite of practically complete involvement of the muscles, the limb may be of some use, provided no deformity has developed; the patient may be able to walk fairly well on a level surface by throwing his leg into hyperextension and locking the joint. The posterior joint capsule and ligaments help to check hyperextension.

Deformities are very likely to occur in a flail-knee. If any of the posterior restraining structures give way, recurvatum develops. Flexion deformity may occur in the early stages, as the result of the posture assumed by the child to secure relief from pain. In the presence of flexion deformity, with some remaining power in the tensor vaginæ femoris or in the outer hamstring, genu valgum is likely to develop. How the presence of deformity interferes with the power of locomotion has already been explained under the sections treating deformity.

Prognosis.—The prognosis of flail-knee is very poor. Treatment, however, should be carefully carried out, for some cases, when handled conscientiously, respond well even at a late date. Some form of apparatus is the most efficient means of dealing with a flail-knee.

Treatment.—The early treatment of flail-knee is the same as that for quadriceps paralysis. Plaster bandages should be applied, and massage and exercise should be carefully carried out. In later treatment, a Thomas ring caliper is very efficient. Drop-catch joints in this caliper enable the patient to keep the leg stiff while walking, and yet permit flexion of the knee while sitting.* Stiffening the knee by arthrodesis is rarely justified.

PARALYSIS AFFECTING THE HIP.

Paralysis in the region of the hip-joint usually occurs in association with severe paralysis of the lower leg. It is seen ordinarily in the bed-ridden or wheel-chair type of patient, or in cases that present little hope of recovery. The condition is often bilateral.

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* For details, see Chapter XIV.

Only rarely are all the muscles about the hip-joint paralyzed; even in severe cases, some fibers of the tensor vaginæ femoris and of the iliopsoas may escape destruction. The intrinsic muscles of the thigh and the gluteal muscles are most frequently involved. Occasionally, an isolated paralysis of the gluteus medius is observed. In rare cases in which the adductors are affected, the hip becomes flexed in the position of abduction, due to the contraction of the iliofemoral band, abductors, and glutei, particularly the gluteus maximus.

Shortening is very likely to occur in hip paralysis as the result of disuse and trophic disturbance of the bone. In some instances, it may be extreme, amounting to 5 inches. The presence of an appreciable amount of shortening often gives rise to a scoliosis, at first postural, but later structural in character.

The prognosis of recovery in paralysis about the hip is very poor. The possibility of the patient's walking depends upon the presence of the iliopsoas and gluteal muscles; if these groups have power, the patient is able to walk by dragging the leg forward, but if they are absent, walking is impossible.

Attempts have been made to replace the power of the gluteal muscles by tendon transplantation.* In properly selected cases the tensor vaginæ femoris has been substituted satisfactorily for the gluteus medius or the gluteus minimus. Lange advocates the substitution of the vastus externus for the gluteus medius, and the transference of the power of the erector spinæ muscle to the greater trochanter in paralysis of the gluteus maximus and medius. It is questionable whether the good results that have been obtained by such transplantations are due to the function of the substituted muscles or to the return of power in the weakened muscles.

Very often the hip paralysis is complicated by extreme deformities of the knee and ankle. In such cases the problem of getting the patient on his feet becomes difficult. It is in this type of case that the ingenuity and skill of the orthopedic surgeon are taxed to the utmost. What a gratifying experience both to the patient and to the surgeon, when a cripple, given up as hopeless, is able to stand and walk a few steps! The patient is enabled to earn his livelihood, and from an economic standpoint is no longer a burden.

The following case illustrates the recovery in a case that was given up as hopeless.

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* For details, see Chapter XIII.

CASE II.—R. L., aged twenty-six years.

History: October, 1917. Seven years previously, this patient had suffered an attack of infantile paralysis. Since that time he had been unable to walk because of deformity in the lumbar spine and hips.

Examination showed a boy lying in bed, with marked lordosis, 45 degrees contracture of the iliofemoral bands, and contraction of the Achilles tendons. There was no contracture of the hamstrings in either leg. The power in both the inner and outer hamstrings of the right leg was good; in the left leg, the inner hamstring had fair power, but the outer hamstring was very weak. The abductors and adductors in both legs were active. The abdominal muscles were weak.

In the right leg, the plantar flexors of the toes and the peronei had good power. The dorsiflexors of the great toe were present, but weak. The gastrocnemius had fair power, the iliopsoas was weak, and the quadriceps had no power. In the left leg, the peronei, the dorsiflexors and plantar flexors of the toes, the iliopsoas, and the gastrocnemius were weak. The quadriceps had no power (Fig. 30.)

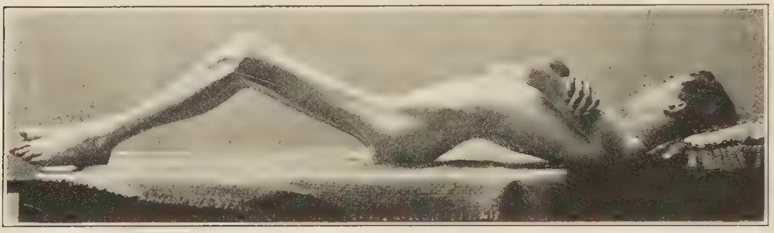

FIG. 30.—Case II. R. L. Hip paralysis. A bed-ridden patient, showing contraction of the iliofemoral bands, lordosis, and contraction of the Achilles tendons.

Treatment: November 3, 1917. Division of the bony attachment of the iliofemoral bands was done.

February 26, 1918, an astraglectomy and tenotomy of the plantar fascia were performed on the right foot, and a tenotomy of the plantar fascia on the left foot.

April 23, 1918, the plaster bandages were removed, and Thomas ring calipers substituted. The braces were equipped with drop-catch joints at the knees, and with stop-joints at the ankles limiting motion to 5 degrees below a right angle.

Result: July 25, 1919, the patient was walking well with braces and crutches. He could sit up in bed, and his back showed only a slight curve. The contraction of the iliofemoral bands had been overcome.

In the right leg, the iliopsoas was weak, and the quadriceps had no power. There was a slight return of power in the dorsiflexors of the toes, especially in those of the great toe.

In the left leg, the iliopsoas was weak, and there was no power in the quadriceps. There was a little motion in the plantar flexors of the toes.

April, 1926. This boy gets about all day, and is at present walking without support of any kind (Fig. 31).

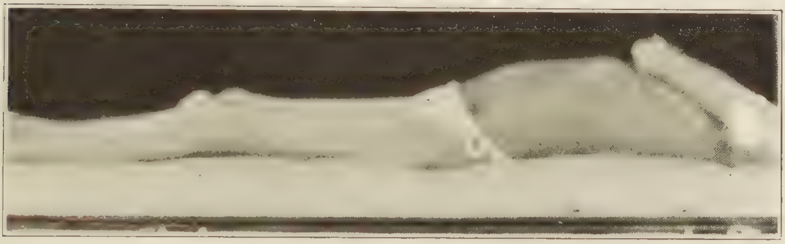

Fig. 31.—Case II. R. L. Hip paralysis. Eight years after correction of deformities. Patient can now walk without support.

FLEXION DEFORMITY.

One of the most difficult deformities with which the surgeon has to deal in infantile paralysis is flexion deformity of the hip, resulting from contraction of the tensor vaginæ femoris, iliopsoas, sartorius, and rectus femoris (Fig. 32). The deformity rarely occurs alone; it is frequently associated with abduction of the hip, flexion deformity of the knee, and talipes equinus of the foot. When such complicating deformities are present, the patient is very crippled and cannot stand erect.

Secondary deformities are likely to develop in connection with flexion contracture of the hip, particularly if the patient is ambulatory. In order to stand erect, the child must tilt the pelvis considerably, thus producing a severe lordosis. Dislocation of the hip, of which further mention will be made, is likely to occur in flexion deformities, especially if an element of adduction is associated with the flexion. In view of the seriousness of these secondary deformities, it is most important to prevent the development of the initial contracture.

Diagnosis.—The diagnosis of a paralytic flexion deformity of the hip is simple. The thigh cannot be extended completely upon the pelvis, and the patient cannot take a standing position without inclining the pelvis and assuming an extreme lordosis. If complicating deformities are present in the knee and foot, the patient is unable to stand upright.

Most surgeons have experienced the difficulty of making a differential diagnosis between an early paralytic flexion deformity of the hip and a case of acute arthritis, or tuberculosis, of the hip. The most confusing symptoms are the limp and pain. In the tuberculous or arthritic hip, there is a history of slow onset, referred pain, limitation of motion, spasm, and frequently, temperature; in the paralytic hip, the onset is sudden, there is usually involvement elsewhere, temperature is rare, and the paralysis is flaccid. Radiograms are of value in making a differential diagnosis between the tuberculous and the paralytic hip.

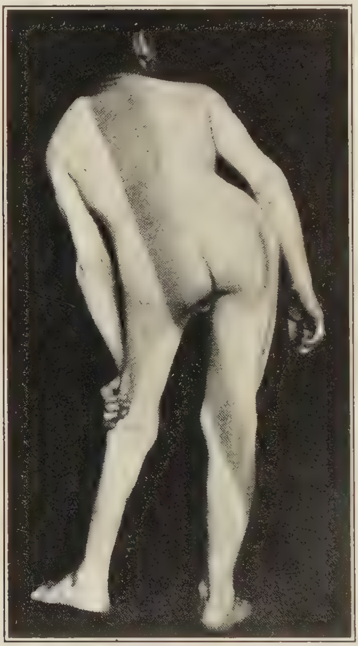

FIG. 32.—Flexion deformity of the hip, with absent quadriceps.

Treatment.—PREVENTIVE AND CONSERVATIVE.—The development of flexion deformity of the hip can be avoided by proper treatment. During the early stage of the disease, the limb should be maintained at rest in proper position by means of a plaster spica, which extends from the rib-line to the knee, or to the end of the paralyzed area if the lower leg is affected (Fig. 33). Shortly after application, the plaster bandage is split to permit local treatment. The spica is worn for a period of several weeks or months, depending upon the severity of the case. When the limb has improved sufficiently, the patient may begin weight-bearing in the spica.

Manipulations are of advantage in preventing contraction. In the convalescent stage, muscle training carried out under proper supervision, and exercise, particularly swimming, are of benefit.

If deformity of the hip has already developed before the patient consults the surgeon, steps should be taken at once to correct it. In mild cases of flexion deformity, the use of traction on the leg, or a manipulation, followed by the application of a plaster spica with the hip in hyperextension, often effects correction. Traction is most efficiently obtained by Buck's extension applied with the leg in abduction. When traction is removed, the hip may be maintained in slight hyperextension by raising the mattress under the pelvis.

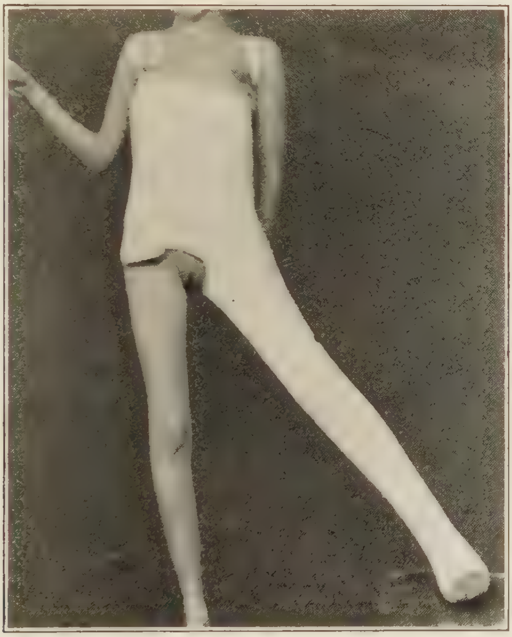

FIG. 33.—Plaster spica.

Any complicating deformities of the lower leg should likewise be corrected. Compensation of shortening is obtained either by means of a cork insert in the shoe or by an extension shoe.

In the convalescent stage, mechanical appliances are of value to maintain the limb in proper position and to assist locomotion.*

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* For details of braces, see Chapter XIV.

In severely paralyzed cases, the Thomas ring caliper transfers the body-weight to the pelvis. When the back as well as the hip is involved in the paralysis, the caliper may be attached to a back support, but such braces are usually too cumbersome to be of any true value.

OPERATIVE*. —When conservative measures fail to correct flexion deformity, operative interference is indicated. Stretching under ether, followed by the application of a plaster spica, may be sufficient to correct early flexion contracture.

Fasciotomy of the tensor vaginæ femoris is indicated when complete correction cannot be obtained by forcible stretching. In order to obtain sufficient overcorrection, care must be taken to follow up the interference with a manipulation. If such precaution is taken, the fasciotomy is attended with good results.

PARALYTIC DISLOCATION OF THE HIP.

Paralytic dislocation of the hip has been extensively studied as to its nature, etiology, and treatment. It is likely to occur in the presence of contractures, particularly when the hip is fixed in the position of adduction. Flexion deformity of the hip, especially when associated with adduction, favors dislocation. The femoral head also is likely to slip out in the presence of marked knock-knee. The relaxation of the joint capsule is another factor that increases the risk of dislocation.

The dislocation may be either partial or complete. In the latter cases, it may be dorsal or anterior; the predominant form is an upward and backward dislocation. When partial, a so-called "snapping hip" may develop.

A dislocated hip aggravates the instability of the limb, and may be painful in walking. The atrophy may be excessive; in some cases the head of the femur becomes practically absorbed.

The diagnosis is made by demonstrating the slipping of the femoral head from its socket, when the leg is in the position of adduction. The elevation of the trochanter above Nélaton's line and the limited abduction are other diagnostic signs. Radiograms furnish the most valuable evidence.

A paralytic dislocated hip has sometimes been confused with a congenital dislocation, but a careful consideration of the features of each condition should make the differentiation simple. In paralytic dislocation, the hip is hypermobile and shows marked atrophy;

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* For details, see Chapter XIII.

paralysis usually is present elsewhere, and there is a history of an attack of infantile paralysis. In congenital dislocation, all the muscles have power, and the history shows that the child has always limped and that he has suffered no disease referable to the hip; limitation of abduction is due merely to the pressure of the hip against the ilium. The trochanter is likely to form a new articulating surface in the congenital type, but it slips about freely in the paralytic type.

Dislocation of the hip may be reduced by the abduction method,* provided all deformities, such as contraction of the tensor vaginæ femoris, have been overcome. After reduction, a spica should be applied, with the leg in extreme abduction. Plaster bandages are worn for a period of three weeks to six months, the abduction being reduced at each change of plaster. An abduction splint may be used after the removal of the last spica.

Reduction by the abduction method may be difficult in old-standing cases, in which the posterior and superior capsule has become extremely relaxed, the inferior capsule contracted, and the head of the femur flattened and atrophied. In such cases, open reduction is advisable and offers the only solution of the problem.†

In selected cases, arthrodesis is justifiable. Such a procedure should be advised only after very careful consideration, not only because of the surgical risk involved, but because in those cases in which it is indicated, concomitant deformities and paralysis are usually so extreme that the stiffened hip is of little aid to locomotion.

FLAIL-HIP.

Flail-hip is a very serious problem. Little power remains in the muscles about the joint. The condition usually is found in conjunction with total paralysis of the lower leg, and it may be bilateral.

If there is any remaining power in the iliopsoas, a caliper brace will assist locomotion. If total paralysis exists, the side involved usually is totally disabled, and little can be done to improve the condition.

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2. Putti: Reported by Mezzari: Chir. d. org. di movimento, 1924, 8, 431.

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* For details, see Chapter XIII.

† Ibid., XIII.

CHAPTER XI.

TREATMENT OF THE UPPER EXTREMITIES.

PARALYSIS OF THE NECK MUSCLES.

The neck muscles are frequently involved in the initial attack, but it is only in rare cases that paralysis of these muscles persists. The residual weakness usually is found associated with paralysis elsewhere, in particular with involvement of the facial muscles.

Involvement of the neck muscles may be so extensive that there is total loss of power. In some cases, activity in one muscle group predominates and gives rise to a slight contraction or torticollis. Such is the case in unilateral paralysis of the sterno-cleido-mastoid.

When the neck muscles have no power, voluntary control of the neck is impossible; the head is wabbly, drooping forward, backward, or to one side. Its most constant position is determined by the muscle group that remains intact.

In torticollis, the head leans toward the healthy side, and the chin faces in the opposite direction. The patient has no voluntary power of turning the head toward the affected side. The absence of the sterno-cleido-mastoid muscle may be determined by testing the patient's power to move his chin downward or to one side against resistance.

Fortunately, the prognosis of paralysis of the neck muscles is encouraging. Many cases respond well to treatment, and the end-results are frequently good.

Enforced rest in bed during the early stage of the disease, with the neck maintained in proper position by means of a plaster helmet or a plaster posterior shell, is most beneficial.

In the later stages, the head should be held in the upright position by means of some form of apparatus.* A Thomas collar is a most satisfactory type of support. If the case is particularly severe, a leather helmet may be used. One of the most important benefits derived from keeping the head properly balanced is the prevention of eye-strain, which so frequently occurs if the eyes are on a different level. Massage, hot fomentations, and muscle training are important adjuncts to treatment by apparatus.

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* For details of supports, see Chapter XIV.

In the more severe types of torticollis, operative interference may be necessary to overcome the contracture of the sterno-cleido-mastoid muscle.

The following case illustrates the recovery of the neck muscles, following conservative treatment.

CASE III.—G. L., aged three and a half years.

Complaint: Paralysis of the right side of the face, of the left arm, neck, and spine.

Duration: Nine days.

Cause: Infantile paralysis.

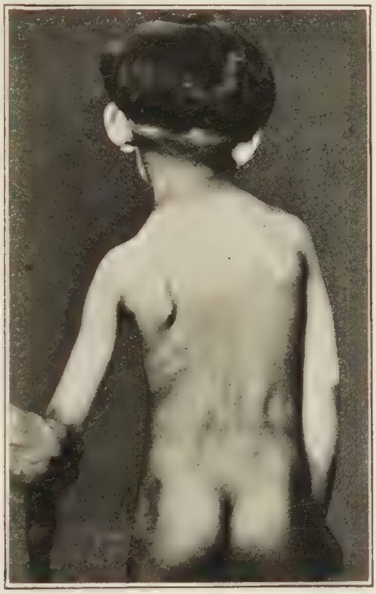

FIG. 34.—Case III. G. L. Neck paralysis. One year, nine months after treatment by plaster bandage and massage.

Treatment: September 14, 1916, a light plaster helmet was applied to the head, neck, left arm, and chest. The helmet was split to allow local therapy.

October 17, 1916, there was no evidence of muscle return.

October 24, 1916, there was slight return of power in the neck and facial muscles.

December 5, 1916, all muscle groups of the arm had regenerated.

March 10, 1917, the power in the neck muscles had returned sufficiently so that only a Thomas collar was needed for support.

March 27, 1917, there was still some facial asymmetry. The muscles of the neck and arm were gradually becoming stronger.

November 6, 1917, the child did not drool any more. He had nearly normal control of the muscles about the face and could maintain the neck and head in good position for short periods. On account of rounded shoulders, a light spring back brace was applied. Setting-up exercises also were advised.

Result: The neck muscles gradually became stronger. Fig. 34 shows the condition *June 18, 1918,* one year, nine months after the onset of paralysis. The muscles continued to regain power, and by *May 20, 1919,* the patient could hold his head erect without a collar.

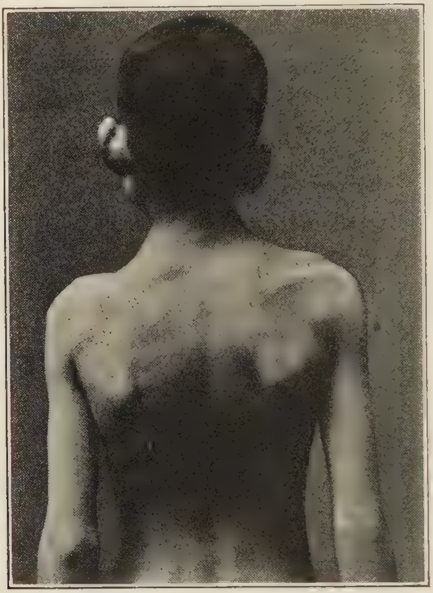

Fig. 35.—Case III. G. L. Neck paralysis. Nearly ten years after treatment.

Fig. 35 shows the condition *April, 1926,* nearly ten years after the attack. The patient now has much better control of his neck and can hold his head erect voluntarily. His back is in much better position.

PARALYSIS OF THE SHOULDER MUSCLES.

The shoulder muscles are not often affected by paralysis. When this region is involved, the extent of the lesion may vary from weakness of the deltoid alone to paralysis of all the shoulder muscles, including the supraspinatus, infraspinatus, rotators, and deltoid (Fig. 36). Paralysis of the deltoid occurs more commonly than affection of the other muscles.

The involvement of the deltoid results from damage to the circumflex nerve. The anatomical changes that take place about the shoulder when this muscle is involved are characteristic (Fig. 37). The deltoid atrophies rapidly. The normal rotundity of the shoulder is lost, and the bony prominences of the acromion and clavicle stand out plainly. The head of the humerus sags and rotates slightly inward. The arm dangles at the side, and only swinging movements are possible. The pull from the weight of the limb causes stretching of the deltoid and joint capsule, and in time leads to stretching of the muscles that were not involved in the original paralysis. The serious effect of a dangling arm impresses upon one the importance of eliminating the weight of the limb as early as possible.

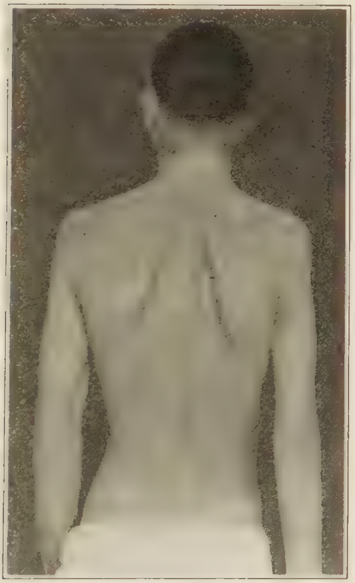

Fig. 36.—Paralysis of the scapular and trapezius muscles.

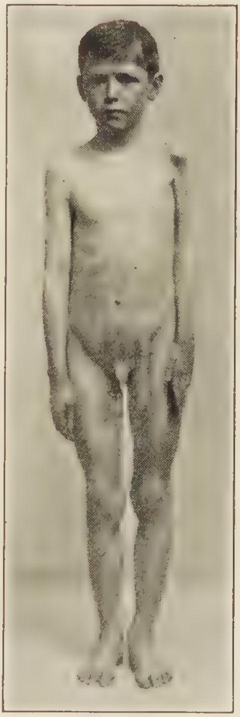

Fig. 37. — Paralysis of the deltoid.

The first step in treatment is to place the arm in a plaster bandage in the position of abduction. The plaster is applied with the elbow at a right angle, the wrist hyperextended, the lower arm externally rotated, and the thumb pointed backward. When the bandage is removed, usually in the course of a few months, an abduction arm splint or leather spica may be used for support.* Hot applications, massage, and passive exercises are of benefit during the period of immobilization. Muscle training, which tends to strengthen not only the deltoid muscle, but also the accessory muscles that perform a function similar to that of the deltoid, should be diligently carried out. If care is taken to prevent stretching of the muscles and joint capsule, and an effort made to restore muscle power by therapeutic measures, operative interference may be avoided in many instances. The success of conservative treatment depends, of course, upon the return of power in the deltoid.

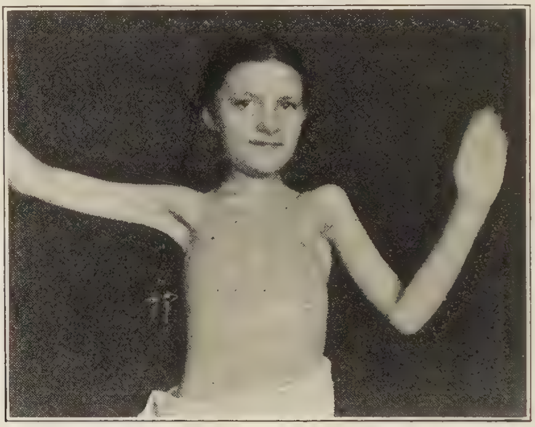

Fig. 38.—Case IV. E. S. Shoulder paralysis, showing attempt to raise arms.

The following case of double deltoid paralysis will serve to illustrate a common result that may be obtained by conservative treatment.

Case IV.—E. S., aged eight years.
Complaint: Paralysis of both arms.
Cause: Infantile paralysis.

Physical Examination: November, 1916. On the right side, the deltoid, triceps, and biceps were weak. On the left side, the deltoid, biceps, and triceps were paralyzed, but showed traces of power. Fig. 38 shows the attempt to raise the arms.

Treatment: November 28, 1916, a plaster bandage was applied with the arms in abduction (Fig. 39). Baths in hot salt water were given daily and massage and passive movements carried out twice daily.

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* For details, see Chapter XIV.

Result: The improvement was slow, but steady.

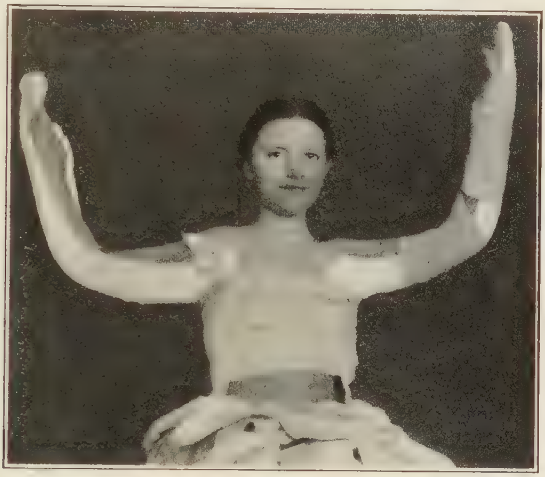

FIG. 39.—Case IV. E. S. Shoulder paralysis. Arms in plaster bandage.

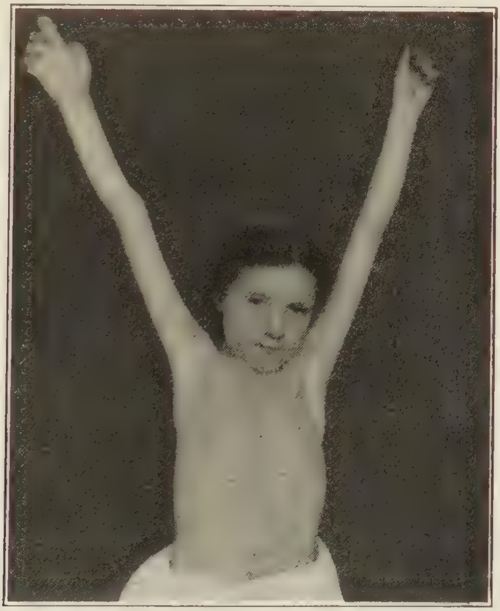

FIG. 40.—Case IV. E. S. Shoulder paralysis, showing return of power in the deltoids following conservative treatment.

May 1, 1917, the right deltoid showed return of power; the left arm had improved, but was still weak.

October 2, 1917, all the muscles of both arms had good power, and the left arm showed excellent function. Fig. 40 shows the ability to raise the arms.

Operative interference is indicated when a flail shoulder-joint develops as the result of complete or nearly complete paralysis of all the muscles of the shoulder region. Arthrodesis of the shoulder gives satisfactory results, provided there is sufficient muscle power present to elevate the scapula, and provided the muscles of the arm have retained enough power so that function in the elbow and hand is good. After an arthrodesis, the arm can be raised in conjunction with the scapula when the trapezius contracts. Not only is a good functional result obtained by arthrodesis, but the cosmetic effect is most gratifying.

In long-standing contractures of the pectorales and teres major and minor, manipulation of the arm into abduction, followed by the application of a plaster bandage with the arm in an abducted position, is indicated. If necessary, forcible manipulation under ether is advisable.

In rare cases of marked internal rotation deformity, an osteotomy may be required.

PARALYSIS AFFECTING THE ELBOW.

Involvement of the upper arm muscles may be due to actual paralysis, or it may be the result of overstretching caused by allowing the arm to dangle. Paralysis of the upper arm muscles is usually found in conjunction with affection of the shoulder muscles.

Regardless of the extent of the paralysis in the region of the elbow, the arm lies flail. The extremity is usually in good position, as the weight of the arm prevents contractures from developing at the elbow. The forearm and hand muscles atrophy quickly, and in some cases the wasting is severe.

The impairment of function depends upon the muscle groups involved. If the triceps alone is paralyzed, the functional power of the extremity is not greatly disturbed, as the weight of the arm accomplishes extension. If involvement of the shoulder muscles is responsible for the flail-arm, the function may be improved by an arthrodesis of the shoulder-joint. But if the biceps or all the flexors of the elbow are paralyzed, function is seriously impaired, since the patient is unable to raise his forearm to his face.

Every effort should be made to restore power to the biceps. As soon as the paralysis is determined, care should be taken to prevent overstretching of the muscle, by maintaining the arm in flexion. If the shoulder muscles also are involved, the arm should be placed in a plaster bandage in the position of abduction, with the elbow at a right angle. When the biceps has sufficiently improved to permit the removal of the plaster, a light leather armlet may be worn.* This support may be arranged to permit free elbow-joint motion, and may be equipped anteriorly with springs to aid the contraction of the biceps.

If the biceps fails to recover under conservative treatment, operative interference is advisable in selected cases. Provided power remains in the wrist and hand, the diamond-plastic operation may be used. If some power of flexion returns in the biceps, which is possible after the arm has been held in flexion, or if the supinator longus takes on the power of flexion, the ultimate result of this operation is good, the patient being able to carry his hand to his mouth. When the power of flexion is lacking in both the biceps and the supinator longus, the skin-flap is liable to stretch under the force of gravity.

Transplantation of part of the triceps muscle into the biceps has been advocated by some operators, but, in the opinion of the writer, such a transplantation is of little value, since the triceps muscle seldom has sufficient power to be of value.

In cases of paralysis with loss of power in all the primary flexors of the elbow, including the biceps, brachial anticus, and supinator longus, but with good power in the flexors of the wrist and fingers, Steindler[1] advocates flexor transposition at the elbow. The common origin of the flexors is raised from the internal epicondyle to a higher level on the humerus. This operation gives the patient only moderate power of flexion at the elbow, but light objects can be carried to the mouth. In cases of combined flail-shoulder and elbow in which arthrodesis of the shoulder as well as the transposition operation is performed, the procedure is of more advantage. The patient can then raise his arm to the horizontal position, and in this position flexion of the elbow is much easier.

PARALYSIS OF THE HAND AND FINGERS.

The hand and fingers usually escape paralysis. When these parts are affected, their involvement often is associated with paralysis of the elbow and shoulder. Since paralysis of the hand is indicative of a lesion of the high motor cells, it is not unusual to see such involvement in fatal cases.

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* For details, see Chapter XIV.

The Distribution of Paralysis.—The extensors of the wrist, fingers, and thumb are involved more often than other muscles of the hand, due to the fact that the musculospiral nerve is affected more frequently than the other nerves of the arm. Not only does paralysis of the extensors occur more commonly than involvement of the flexor group, but it is more severe in character. Steindler,[1] in a study of the distribution of paralysis in the individual hand muscles in 36 cases, found that in the extensor group there were more cases of total than of partial paralysis, while in the flexor group, the reverse was true. Complete paralysis of the extensor group is not always the result of involvement in the initial attack, but is often due to the stretching of muscles only partly paralyzed. If the importance of preventing muscle stretching in the early stage was appreciated, and proper steps taken to avoid it, there would be no further damage to the extensor group, and it is possible that the muscles, having been placed in a position favorable to recovery, might regain power.

Involvement of the musculospiral nerve may occur alone, but it is more usual to find it associated with impairment of the median and ulnar nerves. When all three nerves are affected, the hand muscles, and the flexors and extensors of the wrist are paralyzed. The main disability is the loss of power in the hand muscles, in particular, the power of opposing the thumb to the finger.

The loss of power in any of the muscle groups does not necessarily indicate that the function performed by these muscles is lost, since other groups may take on the action of the paralyzed part. For example, the loss of power in the interossei may be replaced by the combined actions of the extensors and flexors of the fingers. The possibility of the patient's circumventing the lost power by bringing accessory muscles into action must be taken into consideration when muscle tests are made.

Prognosis.—Provided conservative treatment is efficiently carried out early in the attack, the prognosis as to the recovery of power in the hand muscles is good. Unfortunately, the early treatment of paralysis of the hand is likely to be neglected if the paralytic involvement elsewhere is extensive. The lack of return of power in many cases may be attributed to neglect of early care.

When paralysis is residual, there are two possibilities of restoring function. Provided sufficient muscle material is available, tendon transplantation may be used. If the involvement is so extensive that transplantation is impossible, arthrodesis will fix the wrist in good position, and if any deep flexor power remains, will result in an improvement of function. The esthetic value of even a slight increase in function in a dangle-hand warrants any attempt at restoration of power.

Paralysis of the Wrist.—**Paralysis of the Extensors.**—Paralysis of the extensor muscle group of the hand gives rise to a drop-wrist. This is the most common deformity seen in paralytic hands. The power of extension is lost, and the wrist is wabbly. The hand is flexed and falls to the ulnar or radial side, depending upon the distribution of the paralysis. Even in the presence of good flexor muscles, the grip is lost, because the wrist is in the position of deformity. Neither extension of the fingers at the metacarpophalangeal joints nor extension of the terminal phalanx of the thumb is possible. There is marked atrophy of the forearm and hand muscles.

In the early stage of extensor paralysis, a plaster bandage should be applied, with the wrist in slight hyperextension, the fingers slightly flexed, and the forearm rotated externally. If involvement of the upper arm is associated with paralysis of the hand, as frequently happens, the treatment of the extensor paralysis is carried out in conjunction with the treatment of the upper arm. Massage, and active and passive exercises are indispensable to the attainment of a good result.

Conservative treatment should be carried out for at least one year. During this period, the wrist should be maintained in the position of hyperextension, in the early stage by means of a plaster bandage, and later by a hyperextension wrist splint or a wrist and finger splint. Various forms of cock-up splints for the fingers also may be used as indicated.*

When conservative methods are ineffective, or when the deformity is one of long-standing, the problem of improving function becomes a surgical one.

Provided there is sufficient muscle material available in the flexor group, extension of the wrist and fingers may be secured by transplantation. In contrast with paralytic conditions in the foot, the strain of weight-bearing and the effect of gravity need not be taken into consideration in transplantations in the hand. Several methods of transference have been devised, among which are those of Jones, Stiles, and Stoffel. The writer has also worked out a method which is giving good results.†

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* For description of braces, see Chapter XIV.

† For details, see Chapter XIII.

Unfortunately, tendon transplantations can be used in but few cases of hand paralysis, as generally the involvement is so extensive that there is not sufficient material for transference. The alternative is to arthrodese the wrist-joint in a position that is functional as well as favorable to the improvement of the finger motion.* If necessary, tendon transplantations to secure extension of the fingers may be used satisfactorily in conjunction with arthrodesis.

In a totally flail-arm, any attempt to restore function is warranted. Arthrodesis of the shoulder-joint in the position of abduction will often make it possible for the patient to raise his arm by means of elevation and rotation of the scapula; a diamond-flap operation will improve the position of the elbow; and finally, arthrodesis of the wrist-joint will place the hand in the position most favorable to use.

Paralysis of the Flexor Muscles.—When the median nerve is involved, the flexor carpi radialis, the flexor sublimis digitorum, the flexor longus pollicis, the palmaris longus, and the outer half of the flexor profundus digitorum are paralyzed. The loss of flexion of the thumb and index finger causes considerable disability.

In median paralysis, Jones recommends transplantation of the extensor radialis longior, and suturing of the two inactive tendons of the flexor profundus digitorum to the two inner active tendons of the same muscle.†

When the ulnar nerve is involved, the flexor carpi ulnaris is paralyzed. Function may be reëstablished by substituting the palmaris longus and suturing the tendons of the flexor profundus digitorum with some of the tendons of the flexor sublimis (Lange[2]).

Paralysis of the Fingers.—Loss of flexion and extension of the fingers is compensated to some extent by the action of the wrist. The patient with paralysis of the long flexors of the fingers can produce flexion of the middle and end phalangeal joints passively, by extending the wrist. In extensor paralysis, the fingers may be extended by flexing the wrist acutely. The loss of power in the interossei, which control the lateral motion, likewise may be compensated by the combined action of the extensors and flexors of the fingers. Care must be taken in muscle tests to distinguish the true action of the individual muscles. The compensatory power naturally is weaker than the function of the true flexors and extensors, or interossei, and is of little practical value.

Extensor Paralysis of the Fingers.—Tendon transplantations are of value in improving the power in the extensors of the fingers, particularly if the wrist is stable in the position of dorsiflexion. The method outlined by the writer may be used in such cases.*

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* For details, see Chapter XIII. † Ibid.

Flexor Paralysis. The flexors of the fingers are seldom affected. If paralyzed, they recover more frequently than the extensors. When function is seriously impaired, tendon transplantation may assist in restoring power. Several methods of transference have been worked out, among which is that of Stiles.†

Paralysis of the Thenar Muscles.—In involvement of the thenar muscles, the power of opposing the thumb to the fingers is lost. This is an important function of the hand, and its impairment causes serious disability.

If the involvement of the thenar muscles is slight, the loss of power in the abductor and short flexor muscles may be compensated by the action of the adductor and the long flexor of the thumb. Such compensatory motion is more efficient when the short flexor has power. In the majority of cases, the paralysis is so extensive that such substitution of power is impossible, and the thumb lies useless in a position of adduction.

The restoration of thumb motion that will be adequate for practical purposes is possible by a plastic method. Steindler, Ney, and Stiles have worked out operative procedures by which they have obtained good results.‡

Contracture Deformity.—Occasionally, contractures of the wrist and fingers occur, due to the presence of unbalanced muscle power, and to allowing the patient to assume positions favoring the development of contracture. In rare cases the fingers become contracted in flexion. Such deformities generally yield readily to treatment by manipulative methods and proper splintage.

If the muscle balance does not return, tendon transference from the contracted side to the opposite side removes the tight muscle pull and helps to secure correction. The transplantations may or may not be supplemented by an arthrodesis of the wrist-joint.

TRUNK PARALYSIS.

Paralysis of the trunk muscles is now recognized to be of more frequent occurrence than was formerly believed. The involvement is usually asymmetrical and associated with paralysis of the lower extremity on the same side. If both legs are affected symmetrically, or practically so, the damage commonly extends higher on one side of the trunk than on the other, although in some cases the lesion may be almost transverse in its limitations. As paralysis of the lower extremities usually is severe in character, it is likely to attract attention, whereas the involvement of the trunk muscles very frequently is overlooked entirely, or if noted at all, the observation is not sufficiently accurate to demonstrate the asymmetrical loss of power.

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* For details, see Chapter XIII. † Ibid. ‡ Ibid.

Several types of deformity may occur in paralysis of the trunk muscles. The most common is *scoliosis*, which develops when the muscles on one side of the median or sagittal plane, being stronger than those on the other, contract and bend the spine toward the unparalyzed or partly paralyzed side. The rotary movement of the vertebræ very rapidly causes this simple lateral curve to develop into a rotaro-lateral type of curvature, or true scoliosis. When once established the deformity cannot be corrected, and in spite of the most earnest efforts, it may tend to increase with great rapidity.

In rarer cases, *lordosis*, or a round back (kyphosis), may develop as the result of an unbalanced pull when the anterior or posterior spinal muscles are paralyzed. While the sequelæ of these deformities are not so far-reaching as those of a true scoliosis, the conditions are equally difficult to treat.

Inasmuch as all these deformities can be prevented by proper care in the early stages, their development represents lack of attention from the point of view of prophylaxis, that is, the prevention of deformity.

Paralytic Scoliosis.—A rotaro-lateral curvature, in the presence of indications of weakness of the muscles of the trunk or of the extremities, is to be considered paralytic in origin. The lateral deviation is always associated with compensatory or secondary curves, which vary in each case depending upon the existing mechanical force.

Etiology.—The primary factor in the development of a paralytic scoliosis is the same as that in the development of all deformities of paralytic origin, namely, a slight unbalancing of normally well-opposed muscles. When a group of muscles is paralyzed, its unaffected opponent contracts and gives rise to limitation of motion. It is often difficult to determine accurately the muscles involved in the curvature; examination of curves whose convexity is on the right side has shown that the muscles on either the right or left side may be paralyzed.

Paralytic scoliosis is progressive in character. Mechanical changes, rotation, and anatomical fusion of the vertebræ produce a resistant rotaro-lateral curvature. Compensatory curves tend to occur early, particularly in the upper dorsal and cervical regions.

The deformity progresses more rapidly if the patient is erect, although it may develop to an extraordinary degree when the child is recumbent in an ordinary reclining posture on a soft mattress. Once deformity has started, its progression is not retarded by the recovery of the paralyzed muscles.

Paralysis and deformity of the extremities, particularly of the lower extremity, play an important role in the increase of the curvature. Flexion of the leg, either from actual lack of growth, or from deformities of the hip, knee, or ankle, causes a tilting of the pelvis, which influences the development of a scoliosis. Such deformities, however, are merely additional factors in the etiology of a curvature, and should not be considered primary causes of a true paralytic scoliosis.

It is always necessary to differentiate a paralytic scoliosis from various compensatory curves of the spine due to deformities of the lower leg. Shortening, or apparent lengthening of the leg as the result of an equinus deformity, produces a tilting of the pelvis, which is compensated by a lumbar curve in order that the patient can maintain proper trunk balance. It is true that these compensatory curves frequently show rotary deformity as well as a lateral deviation, but they differ from the paralytic scoliosis in that they represent a well-balanced trunk condition and compensate for deformity of a weight-bearing extremity.

Types of Curves.—The first curve to develop is a slight lateral deviation, called in young children the "functional" curve. This deformity may develop without being observed while the patient is lying in bed recovering from an acute illness. Compensatory curves begin to develop very early in the upper dorsal and cervical regions, but they are so slight that they have little influence upon the development of the original curve or upon its treatment.

The progress from a functional curve to a true structural curve, in which the vertebræ are rotated, is rapid. Very often the deformity is well advanced before it is observed, for such curves may develop insidiously, and there may be no symptoms indicating the condition. Once the rotaro-lateral curvature is established, the spine cannot be straightened by any methods in use at present. The importance of preventing the development of structural curves can be readily appreciated.

Elements in the Deformity.—*In the Functional Curve.*—In the early functional curve, the intervertebral discs are compressed on the concave side. If the lateral deviation is allowed to persist, the discs very soon become narrow on this side and assume a wedge-shape. It is to be noted that this fixed deformity is present in the functional curve.

In the Structural Curve.—Shortly after this stage, there begins to be some compression at the edges of the vertebræ themselves, and they, too, gradually assume a wedge-shape. The vertebræ shift to the side of the deformity, at the same time moving somewhat backward and rotating in the direction of the curve. As the vertebra rotates, it carries the rib with it. The weight-bearing line now comes up through the shallow side of the curve, and the edges of the vertebræ on this side become heavier than the outer edges. The vertebræ begin to throw out processes—buttresses of bone—which constitute Nature's attempt to resist advancing deformity. The intervertebral discs are replaced by buttresses of bone, a reaction to the excessive strain under which they are placed. In fact, if these discs remained intact, deformity would develop even more rapidly. The spur formations and the bony processes replacing the soft tissues eventually produce a natural fusion of the vertebræ involved in the curve.

Adaptation of Long Structures, Chest, Soft Tissues, Nerves, Scapula, and Hip.—As the vertebra rotates into the position of deformity, the rib juts backward and loses its normally rounded curve. In very severe cases, the ribs become so sharply angulated that the deformity is called "razor-back."

The chest may be very much restricted, not only in its capacity, but also in its ability to change its shape. Breathing may be almost entirely abdominal. The chest on its lateral aspect is crushed in, making the ribs angular in the posterior aspect. The thickness of the chest in the antero-posterior diameter is very much diminished, and the lateral diameter on the shallow side is sometimes very much increased.

The soft tissues readily adjust themselves to the structural body changes. The alterations in these structures, for the most part, are unimportant. In a severe curve, the torso is so greatly shortened that it affords less space for the soft structures. The abdominal structures easily accommodate themselves to the changes in the abdominal cavity. One observes cases in which the ribs are actually telescoped into the pelvis, and yet these patients have no abdominal symptoms and are apparently well as far as functions are concerned. The lungs also adjust themselves well to the deformity, even when the ribs are actually locked and no expansion is possible. Patients having a lack of respiratory space are poor pulmonary risks, in that they tend to develop tuberculosis more readily than individuals with normal chest expansion. Such patients also are not likely to make a good recovery from acute pulmonary disease.

Impingement or pressure on the vertebral nerves at their exit through the foramen may occur in severe curves with an excessive overgrowth of the buttressing structure. Such pressure gives rise to the symptoms of pain, numbness, or other neurological symptoms.

The scapula on the side of the rotation is elevated and becomes concave. The other scapula is lowered automatically.

In extreme curves, and particularly in those involving the lumbar spine, the hip on the concave side of the curve is apparently higher. Occasionally, there are structural changes between the third and fourth vertebræ, or at the point of impingement of the transverse process of the last lumbar vertebra on the ilium.

Diagnosis.—In extensive cases of paralysis, it is very important to examine the back muscles in order to determine any weakness or paralysis. Examination of these groups is often overlooked when the extremities are severely paralyzed and require attention. At times, it is most difficult to make an early diagnosis of paralysis of the back muscles, because the patient is so ill that it is impossible for him to attempt to flex or extend the spine. In view of this difficulty, the involvement of the back muscles often is not determined until convalescence is well established.

Severe paralysis of the trunk muscles is determined by noting if a functional curve appears when the patient tries to sit up, and by testing his ability to flex or extend the spine in the sitting position. Symptoms of pain in the lateral spine are common. Mild cases of paralysis may not be recognized until the patient becomes ambulatory, and then the parent notices that the child holds one shoulder high, or that one hip is higher than the other, or that the waistline is sunken. The actual deformity of the spine may not be observed.

Scoliotic cases should be examined very carefully. The back must be fully exposed, and the light should fall directly upon it. The eye of the examiner should be opposite the center of the back. The patient stands at ease, with the weight equal on the feet, and the arms hanging at the side. The spinous processes are then marked.

The patient is examined from the posterior, anterior, side, and rotative positions, and the general hygiene, muscle tone, flexibility, and limitation of motions noted. The position and rotation of the scapulæ, the hollowness of the chest and back, the exaggeration of the waist-line, the prominence of one hip, and any inclination of the pelvis are observed carefully. The site of the rotation deformity and its degree can be estimated when the patient bends forward. It is often difficult to determine the degree of motion in paralytic backs, but the function can be estimated roughly by having the patient assume a corrected position when sitting up. Passively, the strength of the muscles may be estimated by assisting the patient in hyperextending the spine from the recumbent position.

Prognosis.—The prognosis depends primarily upon whether or not a case is treated. Untreated cases of curvature never improve, and the deformity always tends to increase (Fig. 41).

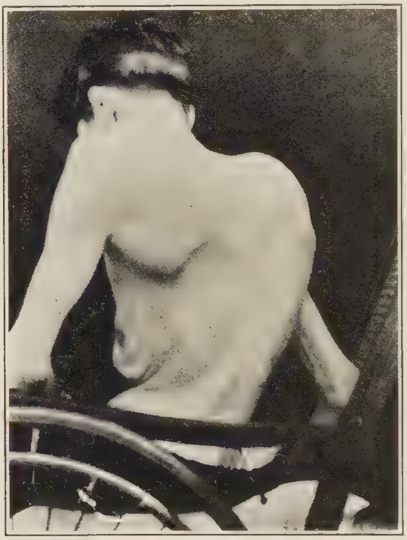

Fig. 41.—A severe structural scoliosis, the result of non-treatment.

The outlook in treated cases depends upon several factors, including:

1. The stage at which the deformity is treated.
2. The type of deformity.

The earlier treatment is begun, the better is the prognosis. If a functional curve is treated properly in its early stages it can be corrected, and provided the affected muscles recover sufficiently, the patient will be able to hold his back in symmetrical position. Unfortunately, it is only in the exceptional case that the muscles regain power. In cases in which the curve has been corrected, but in which unbalance persists because of paralyzed muscles, operative fusion of the vertebræ will ensure permanent stabilization of the back in symmetrical position.

The prognosis of structural scoliosis is unfavorable. Structural curves, regardless of the degree of their severity, cannot be corrected. The increase of deformity in all cases can be prevented by operative fusion of the vertebræ. If the spine is very flexible the curvature may be reduced to some extent, but the process is tedious and of long duration.

Treatment.—PREVENTIVE.—The importance of prophylactic treatment in scoliotic cases cannot be overestimated. If the back is watched carefully from the onset of paralysis, and steps taken to avoid any asymmetry of the trunk, the development of curvature can be prevented.

Prophylactic treatment should begin while the patient is still in bed. The child should rest on a rigid surface, which may be secured by placing a fracture board under the mattress, or by the use of a Whitman frame. The latter is more practical in treating young children.

When the patient sits up, the back should be supported by a light spring back brace or leather jacket.* Frequent examinations should be made to obviate any lateral deviation of the spine. If one side of the body droops, the patient should be kept in the recumbent position for a longer period, and massage and exercises carried out with the hope that the involved muscles will recover.

If no improvement takes place within a reasonable period, it is assumed that the muscles will not recover power. It is in these early cases in which a correct posture cannot be maintained that fusion of the vertebræ by operation is to be particularly recommended.†

CORRECTIVE—Functional curves presage the development of more serious deformity and, therefore, should be corrected immediately upon being observed. A functional curve is always curable. Although deformity is present, it involves only the invertebral discs, which are soft tissue and can be restored to normal position. The important principle in the correction of spinal curves is the same as that in the correction of other deformities, namely, the establishment of overcorrection. Recumbency in hyperextension and stretching by braces and jackets constitute the treatment.

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* For details, see Chapter XIV.

† For details, see Chapter XIII.

When the curve has been overcorrected, operative fusion of the vertebræ will secure fixation of the spine.*

Cases of structural scoliosis cannot be corrected, however mild they may be, but the deformity always can be prevented from progressing, and in some cases the curvature can be reduced. Recumbency in hyperextension, jackets and braces, and finally the Abbott[3] method of forcible correction may be used in the attempt to reduce the curve.

The Abbott method differs from other corrective procedures in that it takes cognizance of the fact that the deformity is in flexion. Previously, the corrective pressure had been exerted with the spine in the extended or hyperextended position, and, according to Dr. Abbott, straightening was impossible because the vertebræ were locked. When the spine is in flexion, the vertebræ are relaxed and can be turned back. Dr. Abbott's principle has created a great deal of discussion, and is not accepted by many students. The writer believes that the Abbott jacket, when applied well, is of value.

The process of reducing a curvature is arduous and tedious, regardless of the method used. Changes in bony structures take place very slowly. A mild case requires active treatment for at least three years, and must be under observation for several years more.

The results are not promising. In many cases we are dealing with vertebræ that are much distorted and even anatomically fused. It is impossible to produce changes in such vertebræ. The exceptional cases in which considerable improvement may be obtained are those of young children with very flexible spines.

Operative fusion of the vertebræ is of value in structural curvature to prevent further progression of deformity, and to obviate the joint strain and arthritic changes to which such cases are subject. While the fusion operation may not result in actual improvement of the curve, the other benefits derived from the procedure warrant its use.

In very severe deformity, Kleinberg's operation,† which combines the technique of Hibbs, Albee, and Forbes, fixes the spine and prevents the increase of deformity.

Kyphosis.—In many cases of trunk paralysis, a round back, or a kyphosis, develops in the early stages. In severe paralysis of the back muscles, the vertebral column droops forward as much as the tension of the intervertebral discs, the ligaments, and the joint capsules permit. When the paralysis of the back muscles is combined with paralysis of the hip muscles, the pelvis falls backward, and the patient has to throw his spinal column forward in order to sit up.

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* For details, see Chapter XIII. † Ibid.

Treatment.—The treatment of this condition consists of recumbency in bed in hyperextension, until the deformity either is corrected or has improved to the greatest degree possible. Massage, breathing and mild setting-up exercises, and braces are used later in securing correction.

When paralysis is complete, a leather jacket will enable the patient to sit up.

Lordosis.—True paralytic lordosis, occurring as the result of involvement of the trunk muscles, is uncommon. Lordosis develops more frequently as a compensatory condition in contracture of the hip. The deformity takes place in the lumbar vertebræ, where the flexibility of the spine is greatest.

When paralytic lordosis does occur, it may be the result of paralysis of the abdominal muscles or of the back muscles. If the back muscles are paralyzed, the trunk tends to fall forward as the result of the unopposed pull of the abdominal muscles. The patient, to avoid falling forward tries to secure a balance between the weight of the trunk and the action of the abdominal muscles, by flexing the trunk into the position of lordosis. In so doing, the pelvis is tilted forward, and the hip-joints are extended.

If the abdominal muscles are paralyzed, the back muscles have no antagonists. The pelvis tilts forward, and the iliopsoas contracts, pulling the lumbar vertebræ into the position of lordosis. In severe deformity the sacrum is almost horizontal.

Treatment.—Supportive treatment is used in cases of lordosis. A light pelvic back brace with an abdominal pad is a most satisfactory support.

Abdominal Paralysis.—Involvement of the abdominal muscles occurs more frequently than ordinarily is supposed (Fig. 42). Paralysis in this region, as in the back, is likely to be overlooked, and is discovered only when the child coughs, cries, or is unable to lift himself. Wickman[4] and Müller[5] found evidence of paresis of the abdominal muscles so frequently in the early stages of poliomyelitis, that they placed paralysis of this region among the important symptoms of the disease. In a series of 64 cases, Müller observed 27 in which the abdominal muscles were paretic or hypotonic. Lovett[6] called attention to the frequent involvement of these muscles.

Abdominal involvement usually is seen in conjunction with paralysis of the muscles of the back, or of the extremities, particularly the legs.

Types of Paralysis.—Mittelstaedt[7] made an extensive study of abdominal paralysis and grouped the most important types as follows:

Paralysis of the recti.
Paralysis of the lateral abdominal muscles.
Combinations of the two groups.

When both recti are affected, the abdomen is arched forward in the region of these muscles. In standing, this arching seems to start with a strikingly marked prominence under the xiphoid process and to bow toward the symphysis pubis.

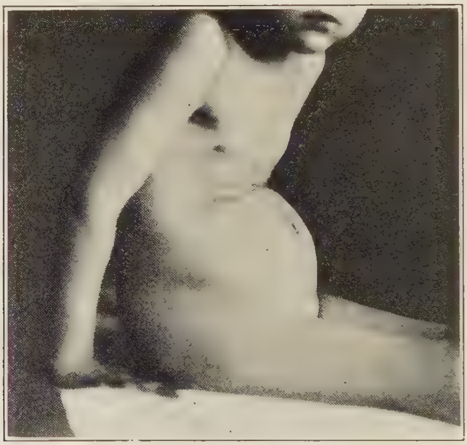

Fig. 42.—Trunk paralysis.

The lateral abdominal muscles are involved more frequently than the recti. In partial paralysis of the obliqui, there is a protrusion of the abdominal wall of the affected part, resembling an abdominal hernia. This distention is due to an active intra-abdominal pressure by gases, which causes a ballooning of the affected muscles. In unilateral paralysis, the pelvis is tilted to the weak side, and the navel is drawn in the direction opposite to the muscles involved.

Combinations of partial or complete paralysis of the rectus and paralysis of the obliqui are seen occasionally. If the paralysis is extensive, the abdominal wall is soft, and there is a tendency for a marked lordosis to develop.

Diagnosis.—When the obliqui are extensively involved, the abdominal wall is felt to be soft and yielding. When the patient coughs or cries, the muscles become distended. These symptoms are more likely to be overlooked than the manifestation of a localized paralysis in which there is a formation resembling a hernia.

Prognosis.—Many cases of abdominal paralysis recover or improve under proper treatment. Permanent paralysis of these muscles, however, is not uncommon. We have already referred to the deformity that may develop in these permanent cases.

Treatment.—In all cases of abdominal paralysis, supportive treatment is indicated, and is best furnished by means of a canvas belt.

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CHAPTER XII.

THE RESTORATION OF MUSCLE POWER.

An attack of poliomyelitis usually results in partial or complete paralysis of one or more muscles. Since the subsequent unbalance will disturb the normal joint function, an attempt must be made to restore power to the affected muscles.

The possibility of recovery of the involved muscles is based, in the main, upon our knowledge of the pathology in the spinal cord. In a typical case of infantile paralysis, the essential lesion is located in the anterior horns of the gray matter of the spinal cord. The motor cells in this area are affected unequally: Some are irreparably damaged, and degenerate completely; others are only slightly injured, and recover spontaneously upon the removal of pressure; and still other cells, which have been damaged, show a slow and gradual recovery. No treatment has any effect upon the muscles controlled by the first group of cells; the muscles governed by the second group recover whether treated or not, as proved by the fact that the residual paralysis is always less than the initial; the recovery of power in the muscles innervated by the third group undoubtedly can be assisted by proper treatment.

Another factor that favors the reëstablishment of muscle power is the columnar arrangement of the motor cells. Since only those in a given area may be affected, it is possible to thrust additional work upon the cells that innervate the muscle from a higher or lower area.

Among the therapeutic measures that aid in promoting the return of muscle power are: Heliotherapy, massage, hydrotherapy, electricity to a limited extent, and particularly reëducation of the muscles, a method known as "muscle training." All these methods are of benefit, and we must rely upon our experience in selecting the measures best suited to the individual case.

In so far as possible, such treatment should be in the hands of a skilled assistant, although a mother who carries out the treatment conscientiously may improve the condition considerably. Therapeutic treatments should be carried out intensively for at least two years.

It is very important to guard against fatigue. It is advisable to exercise the affected muscles too little rather than to risk injuring them by over-use. Excessive stimulation of the nerves, with a consequent over-tiring of the weakened muscles, may delay recovery in the involved area. The writer has noted relapses from over-exercise in cases that were improving. It is often difficult to judge when the point of fatigue has been reached. The patient's endurance will be found to vary from day to day, or even during the same day. Patients with extensive or long-standing paralysis tire quickly.

It is not always easy to prevent muscle fatigue. Particularly is this true in the case of a child who suffers only a slight impairment, and whose general activity is restricted only with the greatest difficulty.

HELIOTHERAPY.

The value of heliotherapy in the general treatment of paralytic cases cannot be over-emphasized. Exposure to the sun not only increases the skin nutrition, but it has an effect upon the deeper structures, with the result that the tone of the muscles is actually improved. Muscles that have had constant sun treatment undergo less wasting and atrophy.

The exposures should be regulated carefully both as to duration and as to the extent of the area exposed. During exposure, the limb should lie first in the anterior half of the plaster shell, and then in the posterior half.

Exposures to the quartz lamp may be substituted for sun treatments. The rays from this apparatus have all the advantages of direct sun exposures. Fifteen minutes' treatment under the quartz lamp is equivalent to an entire day's exposure to the sun. The light exposures can be given regularly, since they are not dependent upon the season or climate. The light treatments should be graduated carefully: The first day, the part is exposed thirty seconds; from then on the time is increased thirty seconds each day until the duration of the exposure is ten minutes; and then the time is increased one minute each day.

MASSAGE.

Massage must not be considered a cure for poliomyelitis. The chief value of massage lies in keeping the muscles in good condition by stimulating the local circulation. If the abdomen is involved, massage helps to increase the peristaltic action of the stomach and intestines and improves the tone of the abdominal muscles. Stroking and deep muscle kneading aid somewhat in overcoming contraction of the strong antagonistic muscle groups.

The best results from massage are secured by an assistant who is trained in the work. Such an operator understands the physiological principles involved and appreciates the effect of each manipulative movement. A trained operator also appreciates the avoidance of fatigue, and regulates the massage accordingly.

Massage should be started as soon as muscle tenderness has disappeared, and treatments should be given daily. The massage period occupies from five to fifteen minutes, depending upon the extent of the pathology. At first, it may be found beneficial to divide the daily treatments into two or three very short periods. It is well to give children short treatments, as they are likely to become irritated by too long a period of massage.

Massage consists of definite manipulations, including stroking, kneading, friction, and percussion. If the whole body is to be treated, the general course of procedure is massage of the legs, abdomen, arms, shoulders, and back. During massage, the limb should always be kept in good functional position. The treatment should be dry in order to secure good contraction of the muscles with a consequent greater flow of lymph, and to increase the local temperature by skin friction.

Treatment by massage should be continued until the circulation is normal and until there are signs of muscular activity. It is often advisable to continue its use during the exercise period as a means of lessening muscle fatigue.

HEAT.

The application of heat, when used in connection with massage, is of advantage. Hot-air baths, by increasing the circulation, raise the temperature of the muscles and put them in the condition most favorable to function. When heat is followed by massage, even better circulation is stimulated, since the massage forces the superficial blood away from the affected part and increases the flow of arterial blood to the weakened limb.

Heat from light rays has been found of more value than heat secured by other means. Electric bakers supplied with many bulbs, which throw an even heat over a large area, are most satisfactory. The limb may be baked from three to fifteen minutes or longer, as required. The temperature should be as high as the patient can stand comfortably. Most patients learn to enjoy the baking.

Moist heat is not so effective as the dry form, because the patient cannot stand water at such a high temperature. In some cases moist heat may irritate the skin.

Warmth is essential to the paralyzed limbs at all times, and the patient should be protected properly against cold by wearing additional clothing, or loosely applied flannel bandages. Constricting bandages should be avoided.

HYDROTHERAPY.

The advantages of hydrotherapy in the treatment of poliomyelitis have been recognized only recently. Hot baths, of ten to fifteen minutes duration, help to relieve pain and tenderness. A lively circulation may be stimulated by alternate hot and cold baths, in which the affected limb first is placed for three minutes under running warm water, whose temperature is gradually increased to just below the burning point, and then is cooled for one minute under running cold water. Whirlpool baths, if properly supervised, have a beneficial effect upon the local circulation.

Hydrotherapy is also of value as a means of eliminating gravity in exercising muscles that are too weak to function under normal conditions. The baths used for this purpose should be made saline, as salt increases the buoyancy of the water. Very often the patient learns to swim before he can walk. When the muscles are sufficiently strong, swimming is one of the most valuable therapeutic measures by which to stimulate function.

ELECTRICITY.

The value of electricity in the treatment of infantile paralysis has long been a subject of discussion. In the past, it has been used extensively to the exclusion of other therapeutic measures. The purpose of this form of treatment has not always been definitely understood, and the indications for its application have not been considered carefully. Undoubtedly, the blind faith that parents had in the use of electricity, and the general impression that it helped in some way to restore power, accounted for much of its popularity.

At the present time electricity is still being used, but its application is more limited. Its advocates now recognize that electricity is only one of several measures of treatment that aid in restoring power to the muscles. They also appreciate the importance of complete functional rest during the early stage, as well as the necessity of preventing the development of deformity.

As yet, no scientific data has been presented that shows wherein lies the value of this measure. The advocates of the use of electricity claim that such treatment, by producing mild contractions, assists in exercising the affected muscles.

The currents used most commonly are the galvanic and the faradic. The galvanic is used to elicit contraction in a *paralyzed* muscle, and it is possible by this means to obtain a reaction in a muscle that cannot be made to contract by any other stimulus. But the question arises as to the value of any form of treatment in paralyzed muscles, whose nerve innervation has been destroyed to such an extent that contraction can be obtained only by direct electrical means. The nerve cells innervating these muscles undoubtedly have been destroyed to such a degree that the muscles will never recover. As to the value of exercising such muscles, with the expectation that they will resume function more easily when nerve innervation returns—it is a well-known fact that provided the paralyzed muscle is maintained in correct position, function will be resumed immediately upon the return of innervation, regardless of whether the muscle has been exercised.

The faradic current is used to stimulate contraction of a *weakened* muscle. Since some voluntary power remains in such cases, functional use of the limb is of more benefit than electricity.

In the opinion of the writer, there is little need of using electricity in the treatment of infantile paralysis. In some cases it may be used to advantage, if correlated with other therapeutic methods, to prevent degeneration and to exercise the muscles when power is beginning to return. Until further proof of its value is submitted, electricity will not be generally accepted as one of the modern methods of treating poliomyelitis.

MUSCLE REËDUCATION.

General Exercise.—Muscles in which some power remains may be trained to assume some of their function by carefully systematized exercise of the part affected, and by general exercise involving the use of the weakened limb. In the opinion of the writer, a correlation of specific and general exercise secures the best return of power. Specific group exercises are beneficial during the period of convalescence between confinement in bed and weight-bearing; general exercise is more effective when the patient is ambulatory. A great deal of emphasis has been placed upon the value of specific group exercises, but very little has been written about the advantages of the general form.*

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* The specific exercises, with indications and limitations, will be discussed further on in this chapter.

Functional use of the muscles is the best stimulation to regeneration, repair, and growth. It benefits not only the region involved, but the general body musculature. Normal activity stimulates the sluggish circulation. The sense of equilibrium, so frequently impaired by an attack of paralysis, is restored more quickly by getting the patient on his feet as soon as possible.

Two factors must be borne in mind in advocating active use of the weakened muscles: The prevention of deformity, and the avoidance of fatigue. The patient should wear some form of supportive apparatus to prevent an unopposed muscle pull which might result in deformity. A careful study of the power of the muscle, to determine just how much work it can stand, will enable us to restrict activity before the point of fatigue is reached.

The time when a patient may begin weight-bearing depends upon the severity and extent of the paralysis. In a mild case, such as involvement of one muscle group of the foot, the child may get up after a few weeks of therapeutic treatment, provided the limb is protected against deformity. In more severe and extensive paralysis, the period of conservative treatment covers several months. Exercise, without weight-bearing, should be continued until the muscles have gained sufficient strength to withstand the strain upon the involved groups. Care should be taken not to get the patient up too soon, because of the danger of fatigue, and the possibility of the development of secondary deformities from static influences and poor habit postures. The surgeon, ever keeping these factors in mind, may judge when the patient is ready to begin weight-bearing. In general, walking with or without the support of crutches is possible during the first six months.

When sufficient strength has returned, sports, such as rowing, golf, riding, and particularly swimming, afford a means of obtaining general exercise. That sport should be selected which will be most beneficial to a given condition. The effect of such exercise is more lasting than that of specific training under supervision. General exercise also involves less expense and less time, and is more likely to be carried out over a longer period.

Specific Exercise.—*Objective.*—Muscle training attempts to establish the return of muscle balance by strengthening the affected muscles and by hypertrophying the remaining strong groups. It is in the latter process that our greatest hope of restoring balance lies.

By continued use of the muscles of the involved area, an attempt is made to increase and perfect any remaining power to such a degree that the muscles will be able to accomplish their movement with nearly normal ability. Just as the expert baseball pitcher, by repeated practice, is able to bring into play every muscle fiber needed to deliver his ball at an exact spot, so the paralytic patient strives to train each muscle to perform its work toward the accomplishment of the customary movement.

Age.—The age of the patient is an important factor, for the executing of a movement depends a great deal upon the capability of concentration. The older child, who can appreciate the objective in view, is able to concentrate his attention upon the attempt to accomplish a given movement. The younger child does not have this power of concentration, and some means must be devised by which the desired motions can be elicited. The introduction of the play spirit is important in such cases. Many children will move their limbs freely when they can splash around in water. Diagrams assist the child in performing his exercises; for example, a circle may be used to outline the movements of the arms. It is often necessary to vary the schemes from day to day to avoid irksomeness and to stimulate the patient's interest.

The child under two years of age presents a very definite problem, as no coöperation can be expected. All kinds of indirect methods must be used to bring the muscles into play. By tickling or pricking the sole or dorsum of the foot, the supervisor can make the child draw his foot upward, downward, or sidewise. By pricking the thigh, he can elicit adduction, abduction, flexion, or extension. By scratching the top of the child's head, or holding up toys or food, the assistant can induce him to elevate his arm or grasp at the object with his hand. Many children thirteen or fourteen months old can understand such words of command as "up," "down," "in," and "out."

Regardless of the device by which the movements are elicited great care should be taken not to sacrifice *accuracy*. More harm than good results from exercises carelessly carried out, in that the already weakened muscle is stretched and the development of contracture in the over-acting antagonist is favored.

Prognosis.—What may we expect from muscle training? In cases of moderate paralytic involvement, we may anticipate a more or less complete recovery. Even in the practically hopeless class in which a very large part of the body is paralyzed, muscle training makes for greater efficiency of the nerve and muscle action, thus improving the body statics so that balance is more nearly normal. Patience, perseverance, and unlimited optimism are indispensable factors in securing good ultimate results.

The prognosis depends to a certain extent upon the patient's possession of a good sense of movement (kinesia). Better results may be expected when the child has a good motor intelligence. It is often noticed that an exceedingly bright child may possess little capability of controlling his own movements, whereas a child somewhat dull and stupid may have a high motor intelligence.

The greatest return of power takes place during the first three to six weeks. Later, the rate of recovery is slower, although partly paralyzed muscles may continue to improve for an indefinite period.

Examination of the Muscles.—A knowledge of the contractile power in each muscle group is an essential prerequisite to treatment. The degree of power present is determined by testing the capability of the muscles to perform their normal movements. The conditions under which such tests are made vary according to the amount of power in the muscle. The first step in the examination is to determine if the muscle can perform its movement, when it is in a position favorable to contraction and working against the force of gravity. The failure of the muscle to react under such conditions does not signify that it is without power. A response may be elicited in a muscle that shows less than normal power, by making the conditions under which it functions more conducive to contraction. The surgeon may detect power in the muscle by assisting in its movement. Gravity may be eliminated either by resting the limb on a table or by immersing it in saline water. A trace of power, which was hitherto not discernible, is often noted when the child is immersed in hot saline water. The power in a very weak muscle may be tested by examining the power of contraction when the limb is at rest.

The power present in a muscle should be carefully graded according to the following classification:

Gone: No evidence of contraction.
Trace: Evidence of slight contraction, with no power to perform any function.
Poor: Cannot overcome gravity, but can perform function with gravity removed.
Fair: Cannot overcome resistance, but can overcome gravity.
Good: Can overcome resistance, but shows no further power.
Normal: Can perform a normal strength test. For instance, the normal quadriceps can lift the body weight from the crouching position.

Some muscles will show power clinically, but it is not sufficient for practical purposes. Take, for example, a case of slightly weakened peronei. They may show an apparently normal contraction clinically, but when the patient stands, the muscles are not strong enough to hold the foot in position, and it goes into the deformity of varus. To determine the usefulness of the muscle more accurately, it should be tested under conditions less favorable to its contraction. Resistance may be supplied by the surgeon's hand, or the limbs may be placed in a position that tends to make the execution of movements more difficult. The final test is to estimate the relation of the power to function and weight-bearing. For example, as a final test of the plantar flexors of the ankle, the patient should stand with the weight on the affected leg, the knee on the well side bent, and from this position should attempt to rise on the ball of the weak foot.

The regular tests cannot be used in young children. Examination may be made by watching their voluntary movements, or by tickling or pricking an extremity with a pin.

After muscle training has been started, frequent examinations should be made to record the improvement. It is often of help to estimate the power of the affected muscle by comparing it with that of the corresponding muscle on the opposite side.

A test that has proved of much value, not only in examination of muscles, but in recording the progress of recovery, is the spring balance test devised by Lovett and Martin. By this method, the quantitative value of the partially paralyzed muscles may be estimated. The technique of the test is described by Lovett and Martin[1] as follows: "The method is designed to test, under conditions of constant position and leverage, by a series of spring balance pulls, the power of the muscles which govern the movement of the limbs. The value of the test consists in the possibility of duplicating exactly the conditions of the first test at succeeding ones, so that a definite idea of gain or loss in muscular strength can be registered in pounds. It is applicable for all tests of power in normal muscles, for determining loss or gain in power at stated intervals, for the determination of the degree of initial weakness in paralyzed muscles, for determining the relative strength of the different muscles before tendon transplantation and similar operations. It has been applied for one year in consecutive tests varying in frequency from ten days to three months to infantile paralysis cases. The result has been an accurate register of general gain and occasional loss in the cases under treatment. The record has the advantage of representing concisely, in figures, the results of detailed muscular examination, and of presenting at later examinations the initial and intermediate conditions of the case.

"The method aims at pulling against a fixed position assumed by the patient, rather than attempting to have the patient initiate a movement, because it was found with children that they would hold a fixed position much more readily than they would initiate a muscular movement.

"The accuracy of the test depends upon the training of two persons, an operator and an assistant, to coördinate the pull of the muscle and the registration of the pull on the scales, and upon the maintenance with exactness of the positions and leverage relationships outlined individually below. Accurate spring balance scales (No. 5) are used of four sizes: 1 to 4 pounds, graded in ounces; 1 to 30 pounds; 1 to 50 pounds, and 1 to 100 pounds. The readings are taken to the half pound, except on the ounce scale. The operator in general controls and maintains the correct position of the subject, stimulates the subject to innervation, braces and guides the limb tested, and calls the moment of give in the muscle tested through watching the action of the muscle itself. The assistant makes the pull along lines accurately determined, beginning and stopping under the direction of the operator. The same command directs the muscular pull of the patient and the scale pull of the assistant. In all cases where the position of the assistant makes this possible, the scale reading is taken by him at the moment when the yielding in the muscle is called by the operator. Except under special circumstances, plantar flexion is the only reading which the operator is required to make.

"Twenty-two readings are taken, for each of which the best position of the subject for the accurate reading of the scales and for constant leverage in limb action has been determined experimentally. The order in which muscles are tested is immaterial except under conditions of weakness, but it is best that the order be constant so that all tests duplicate each other as completely as possible." The apparatus required for the test is shown in Fig. 43.

The application of the test to elicit plantar flexion will serve to illustrate its use.

"The subject lies on his back on a smooth table. The foot is braced against a 3 to 1 lever (No. 1). The scale hook is inserted in the ring of the lever upright. The lever must be adjusted so that the ball of the foot in maximum plantar flexion rests squarely upon the lever pad (No. 2), with the upright at an angle of from 60 to 80 degrees to the table. The lever is held in position by C-clamps (No. 3). The pull is made by the assistant from the head of the table with the scale horizontal and in line with the leg being tested, and is increased in intensity to the point where the muscular resistance is overcome. To prevent slipping on the table, the shoulders of the subject are held by the hip braces (No. 4). The muscle gives at about 45 degrees of plantar flexion with a rather sharp break in the resistance offered to the spring balance. All measurements of degrees are made to the plane of the table unless otherwise specified. The operator guides the position of the foot, stimulates the patient to innervation, and calls the moment of break in the muscle to the assistant for reading, or reads the scale himself. The reading of the scale must be simultaneous with this break."

Indications.—Muscle training may be started when we are reasonably sure that the inflammatory process has ceased, and when the patient can move his limbs freely without pain. The initiatory time varies from three to eight weeks after the attack. All pain, sensitiveness, tenderness, and pyrexia should have subsided before muscle training is begun.

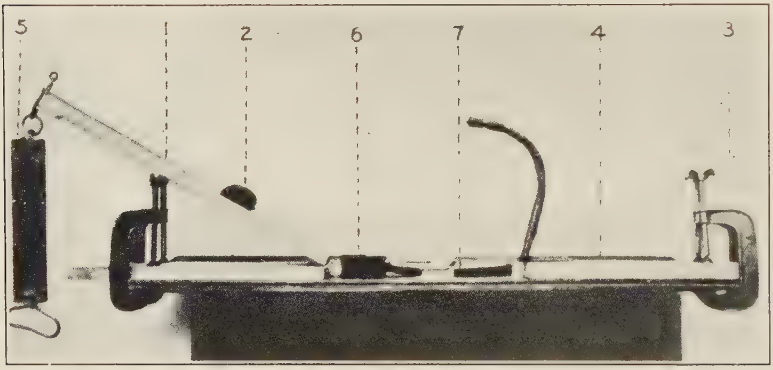

Fig. 43.—Table and apparatus used in muscle testing. (Lovett.)

Regulation of the Exercises.—It is best for the supervisor to be alone with the patient during the exercise period. The whole attention of the child is required to obtain the best results, and the presence of another person, with possibly the exception of the mother, is distracting. Muscle training in group formation is not satisfactory. The writer has observed children exercising in groups, who, not heeding or understanding the exercise being given, performed movements that actually tended to favor the development of deformity.

If possible, the clothing should be removed during the treatment, not only to prevent hampering the action of the muscles, but also to permit the instructor to observe the muscle contractions accurately. If the patient is an older child, simple garments such as those shown in the illustration (Fig. 44) are most practical. The instructor should never guess at the effect of the exercise, for actual damage may be done if the weakened muscles are not observed closely.

After the exercise period, the limbs should be kept warm. They also should be rested in correct anatomical position, support being supplied by some form of apparatus if necessary.

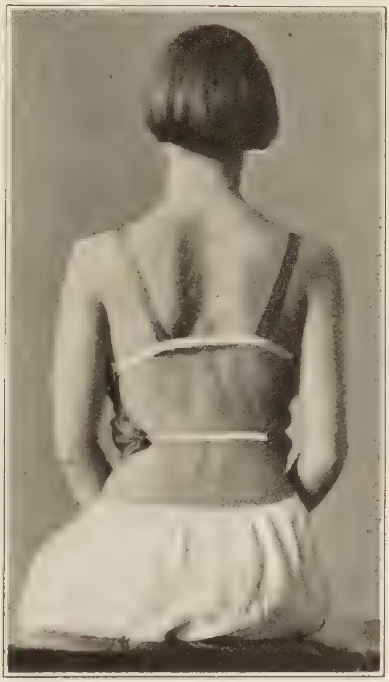

FIG. 44.—A simple form of garment which permits good observation.

The duration of the exercise period depends upon the condition of the muscles involved. The first few periods should be short, and the exercises performed very gently. As the strength increases, the treatments may be more strenuous and of longer duration. To obtain the full benefit of muscle exercise, the movements should be performed daily, six days a week. As soon as the condition permits, each exercise is carried out ten or twelve times, care being taken that it is performed accurately each time, and that the rhythm is slow enough to permit a few seconds of rest between each movement.

It is evident, from the many requirements necessary for the effective application of muscle training, that the treatment must be supervised by one who appreciates the principles underlying this work. The instructor must have an accurate knowledge of the anatomical and physiological principles involved. He must be able to determine if the proper muscle is functioning when certain movements are elicited; otherwise, there is a possibility that the child will circumvent a weakness by using the already strong muscles, and thereby put additional strain upon the weakened parts. A trained instructor appreciates the importance of fatigue, whereas parents or other attendants may allow over-activity of the muscles. A pleasing personality, ingenuity, and a certain amount of enthusiasm are other qualifications that help the supervisor in eliciting response in the patient.

It is well to start the exercise period by working up the general circulation by breathing exercises. The normal individual obtains plenty of stimulus from his daily exercises to keep his circulation lively. The paralyzed individual cannot be active enough to secure natural stimulation, and the circulation must be worked up artificially. Breathing exercises, which fill the lungs with fresh air, are effective in this respect. The number of breathing exercises and the amount of effort put into them will vary according to the condition of a given case, and must be regulated by the instructor. The technique is as follows:

1. The patient lies on his back. The instructor draws his arms upward and downward slowly, under as much voluntary inhalation and exahalation as the patient is able to command.

2. The patient, lying on his back with his knees in a comfortable position, his arms thrown back in a "half-bent" position, and his elbows lowered, is urged to inhale as deeply as possible, and then to exhale slowly. The patient may also experience a good chest expansion by lifting and spreading the lower ribs with his fingers at the time of inhalation. This breathing exercise expands the chest, stretches the spine, and benefits the muscles of the chest and upper back.

3. The patient lies on his back with his knees up. The right arm is thrown backward in the "half-bent" position, and the left hand is placed on the abdomen. The patient inhales deeply, lifting the abdominal wall at the same time, and then exhales. This abdominal breathing is a simple but effective exercise which benefits the abdominal muscles.

The specific exercises for each muscle group are based upon the movement that the muscle ordinarily performs. Of necessity, they must be graduated according to the degree of power present. If the muscle is very weak, the supervisor must assist in the exercises, and the limb must be placed in a position favorable to the contraction of the muscle. If necessary, gravity is eliminated by resting the limb on a table or immersing it in saline water. Passive exercises are carried out until voluntary power returns. When the patient is able to accomplish a movement under more difficult conditions, resistance is supplied by gravity, by hand, or by some form of apparatus.

In performing the exercises, the patient should always start with the limb in correct anatomical position. Not infrequently a child is seen to begin his exercises from a position of deformity, and after completing the movement, to bring his feet to rest again in improper position.

Care must be taken not to confound the power in one muscle with that of another, and not to mistake the action of accessory muscles for that of the muscle being treated. For instance, when the arm drops to the side, the slow relaxation of the active biceps may be confused with the contraction of the triceps. To obviate such confusion, the arm should be flexed at the elbow and held on a level with the shoulder, and the patient should try to extend his arm from this position. Then again, in biceps paralysis, when the patient attempts to flex his arm, the contraction of the supinator longus and the brachialis anticus may be mistaken for that of the biceps.

Included in the following list are the normal movements and tests of each muscle, performed under conditions favorable to contraction and working against the force of gravity. It is upon these movements that the exercises are based, care being taken to graduate them according to the power in the muscle, as explained above. For the compilation of the muscles performing each function, as well as for some of the tests, Lovett's *Treatment of Infantile Paralysis* has been consulted.

The Toes.

Flexion.

Flexor muscles of the toes:

Flexor longus digitorum, four toes (acting on both joints).
Flexor brevis digitorum, four toes (acting on the first joint).
Flexor accessorius, four toes.
Flexor longus hallucis, big toe.
Flexor brevis hallucis, big toe.
Flexor brevis minimi digiti, one toe.
Lumbricales, four toes.
Interossei, three toes.

Muscle Action.—This group of muscles flexes the toes toward the sole of the foot, as if they were gripping an object.

Test.—The patient lies or sits, and bends his toes toward the sole of the foot.

EXTENSION.

Extensor muscles of the toes:

Extensor longus digitorum.
Extensor brevis digitorum.
Extensor proprius hallucis.
Interossei.
Lumbricales (Cunningham).

Muscle Action.—This group draws the toes up toward the dorsum of the foot.

Test.—The patient lies or sits, and bends his toes toward the dorsum of the foot.

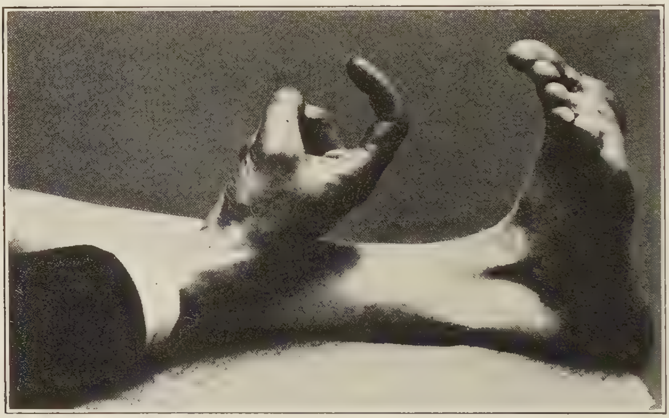

FIG. 45.—Test for dorsiflexion of the ankle.

THE ANKLE.

DORSAL FLEXION.

Dorsal flexor muscles of the foot:

Tibialis anticus.
Extensor longus digitorum (Piersol).
Extensor communis digitorum (Cunningham).
Extensor proprius hallucis.
Peroneus tertius.

Muscle Action.—This group dorsiflexes the foot toward the leg.

Test.—The patient tries to flex his foot toward the examiner's finger which is held slightly up the leg (Fig. 45).

PLANTAR FLEXION.

Extensor muscles of the ankle:

- Gastrocnemius.
- Plantaris.
- Soleus.
- Tibialis posticus.
- Peroneus longus.
- Peroneus brevis.
- Flexor longus digitorum.
- Flexor longus hallucis.

Muscle Action.—This group extends the foot on the leg.

Test. The patient tries to extend the foot toward the examiner's finger, which is held slightly away from the great toe.

INVERSION OF THE FOOT.

Muscles which invert the foot:

- Tibialis anticus.
- Tibialis posticus.
  - When the anterior tibial muscle is paralyzed, the extensor hallucis has slight power to invert the foot.

Muscle Action.—This group turns the foot inward.

Test. The patient attempts to turn the foot inward toward the surgeon's finger, which is held slightly above the great toe on the inner border of the foot.

EVERSION OF THE FOOT.

Muscles which evert the foot:

- Peroneus longus.
- Peroneus brevis.
- Peroneus tertius.

Muscle Action.—This group turns the foot outward.

Test.—The patient is requested to evert his foot toward the examiner's finger, which is held slightly away from the extremity just above the ankle.

THE KNEE.

FLEXION OF THE KNEE.

Flexor muscles of the knee:

- Biceps.
- Semitendinosus.
- Semimembranosus.
- Gastrocnemius.
- Popliteus.
- Sartorius.
- Gracilis (Cunningham).

Muscle Action.—The flexor muscles bend the knee on the thigh.

Test.—The patient, lying on his face, with his leg straight, flexes it until the heel touches the buttock (Fig. 46).

EXTENSION OF THE KNEE.

The extensor muscle of the knee is the quadriceps extensor.

Muscle Action.—This muscle extends the knee.

Test.—When the muscle has normal power, the patient, sitting on a table, can extend the leg from the flexed position.

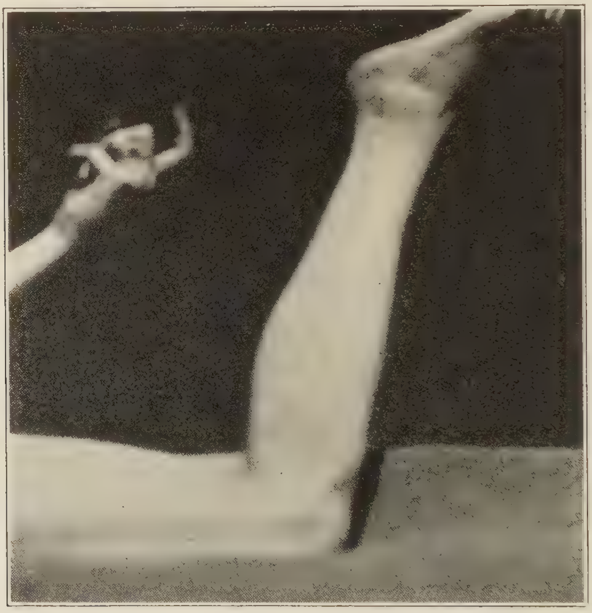

FIG. 46.—Test for flexion of the knee.

THE HIP.

FLEXION OF THE HIP.

Flexor muscles of the hip:

- Sartorius.
- Psoas major and iliacus.
- Rectus femoris.
- Pectineus.
- Adductor longus.
- Gracilis.
- Obturator externus (Piersol).
- Adductor brevis (Cunningham).

Muscle Action.—This group flexes the thigh on the trunk.

Test.—The patient, lying on his back with his leg straight, raises the leg from the table (Fig. 47).

EXTENSION OF THE HIP.

Extensor muscles of the hip:

Gluteus maximus.
Gluteus medius (Cunningham).
Gluteus minimus (Cunningham).
Adductor magnus (Cunningham).
Hamstrings.

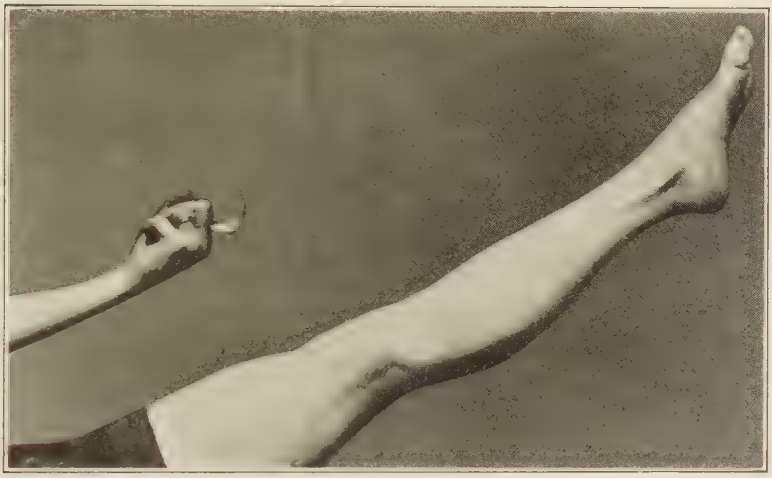

FIG. 47.—Test for flexion of the hip.

Muscle Action.—This group extends the thigh on the trunk.

Test. The patient, lying on his face with the knee straight, can hyperextend the leg.

INWARD ROTATION OF THE HIP.

Muscles that rotate the hip inward:

Tensor fasciæ latæ.
Gluteus medius (anterior fibers).
Gluteus minimus (anterior fibers).
Semimembranosus (Piersol).
Semitendinosus (Piersol).
Gracilis (Piersol).
Iliopsoas (Piersol).

Muscle Action.—This group of muscles rotates the thigh inward.

Test.—Under normal conditions, the patient, sitting with the knees flexed and the lower legs hanging over the edge of the table and held firmly by the surgeon, can move the heel of the affected foot away from the other foot, thus rotating the hip inward.

OUTWARD ROTATION OF THE HIP.

Muscles that rotate the thigh outward:

Gluteus maximus (lower fibers).
Gluteus medius (posterior fibers) (Cunningham).
Gluteus minimus (posterior fibers) (Cunningham).
Quadratus femoris.

During extension:

Pyriformis.
Gemelli.
Obturator externus.
Sartorius.
Iliopsoas (Cunningham).
Pectineus.
Adductor longus (Cunningham).
Adductor brevis (Cunningham).
Adductor magnus (Cunningham).
Biceps femoris.
Obturator internus.

Muscle Action.—This group of muscles rotates the thigh outward.

Test.—Under normal conditions, the patient, sitting with knees flexed and the leg hanging over the edge of the table, can move the foot of the affected leg inward across the other leg, thus rotating the thigh outward.

ABDUCTION OF THE THIGH.

Muscles that abduct the thigh on the trunk:

Tensor fasciæ femoris (Cunningham).
Gluteus medius.
Gluteus minimus.
Obturator externus (Cunningham).

During flexion:

Gemelli.
Pyriformis.
Obturator internus.
Sartorius.
Gluteus maximus (upper fibers) (Cunningham).

Muscle Action.—This group of muscles abducts the thigh on the trunk.

Test.—Under normal conditions the patient, lying on the well side, can abduct the thigh by raising the affected leg with the knee straight and in line with the body (Fig. 48).

ADDUCTION OF THE HIP.

Muscles that adduct the hip:

Adductor longus.
Adductor brevis.
Adductor magnus.
Pectineus.
Gracilis.
Quadratus femoris (Cunningham).
Gluteus maximus (lower fibers) (Cunningham).

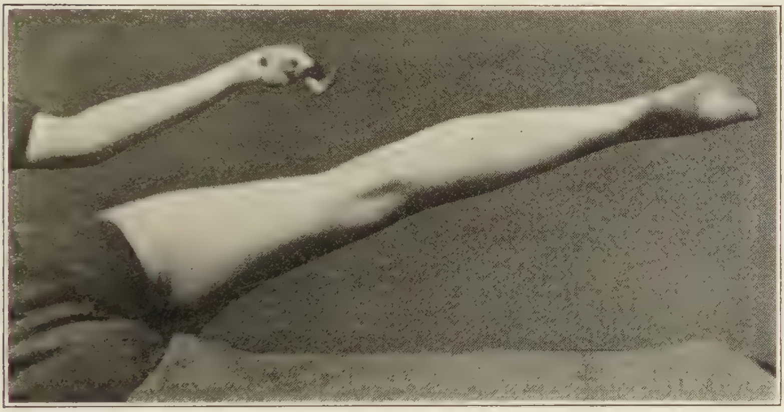

FIG. 48.—Test for abduction of the thigh.

Muscle Action.—This muscle group adducts the thigh from the mid-line.

Test.—Under normal conditions the patient, lying on his back with the affected leg in extreme abduction, can bring the leg in toward the other leg.

THE SPINAL COLUMN.

FLEXION OF THE SPINE. (FORWARD BENDING).

Flexor muscles of the spine:

Rectus abdominis.
Pyramidalis (Cunningham).
Obliquus externus.
Obliquus internus.
Transversalis abdominis (Cunningham).
Psoas major and minor.

Muscle Action.—This group bends the body forward.

Test.—The patient lying on his back can rise to an upright sitting position (Fig. 49).

EXTENSION OF THE SPINE.

Extensor muscle of the spine (Cunningham):

Erector spinæ.

Muscle Action.—This group, functioning with the extensor muscles of the thigh, bends the body backward.

Test.—The patient lies face down with the legs held by the surgeon. From this position the child can bend the spine backward, raising the head and the whole body clear of the table.

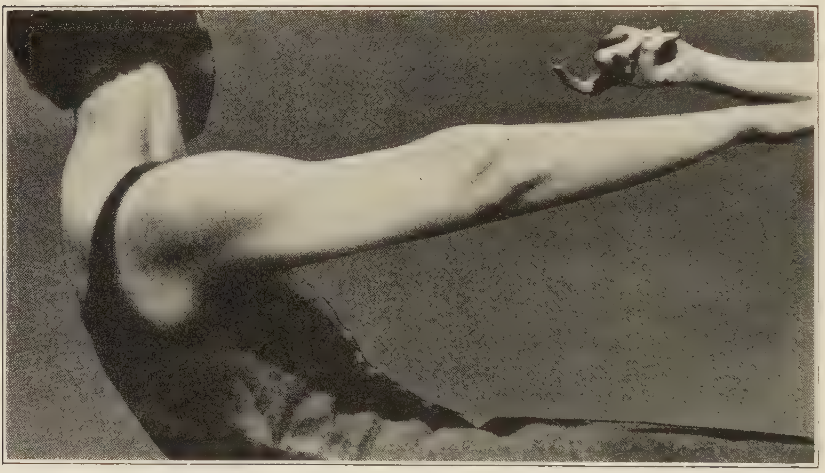

FIG. 49.—Test for flexion of the spine.

LATERAL FLEXION OF THE SPINE. (SIDE BENDING). (This movement is not clearly separated from the movement of rotation of the spine.)

Lateral flexor muscles of the spine:

Rectus abdominis.
Obliquus externus and internus.
Transversus abdominis.
Psoas major and minor.
Quadratus lumborum.
Erector spinæ.

Muscle Action.—This group of muscles bends the body to the side.

Test.—The patient lies on the well side with the arms folded and the legs held down by the surgeon. From this position the patient can lift his body from the table.

ROTATION OF THE SPINE. (This movement is not clearly separated from that of lateral bending of the spine.)

Rotator muscles of the spine:

Erector spinæ.
Obliquus externus and internus.

Muscle Action.—This group twists the body to one side.

Test.—The patient, sitting with his arms folded and the hips steadied by the surgeon, twists the body to one side.

THE NECK.

In the neck movements many muscles are involved, and there is only partial agreement among anatomists as to their particular function. The list of muscles, therefore, given in this connection is of little value.

FLEXION OF THE HEAD.

Flexor muscles of the head:

Sterno-cleido-mastoids (Piersol).
Omo-hyoids.
Mylo-hyoids.
Sterno-hyoids.
Sterno-thyroids.
Recti capitis antici.
Longus colli.
Other smaller muscles.

Muscle Action.—This group brings the head forward on the neck.

Test.—The patient, lying on his back with the shoulders held down, raises the head from the table.

EXTENSION OF THE HEAD.

Extensor muscles of the head:

Trapezii (clavicular) (Piersol).
Splenii capitis.
Recti capitis postici.
Obliqui inferiores.
Other muscles.

Muscle Action.—This group bends the head backward on the neck.

Test.—The patient, lying face down with shoulders held firmly, can raise the head.

LATERAL FLEXION OF THE HEAD.

Muscles that flex the head laterally:

Sterno-cleido-mastoid (the most important).

Muscle Action.—This group bends the head to one side or the other.

Test.—The patient, lying on the unaffected side, attempts to raise the head laterally from the table.

ROTATION OF THE HEAD.

The muscles that rotate the head. Many muscles produce this movement, including the:

Sterno-cleido-mastoid.
Splenius capitis.
Longissimus capitis.
Semispinalis capitis.
Obliquus capitis inferior.
Rectus capitis posterior major and minor.

Muscle Action.—This group turns the head from one side to another.

Test.—The patient turns the head from one side to the other.

THE SHOULDER.

FLEXION OF THE HUMERUS.

The muscles that flex the humerus to the horizontal line, raising the arm forward in the antero-posterior plane:

Deltoid (anterior fibers).
Pectoralis major (clavicular).
Biceps.
Coracobrachialis (?).

Above the horizontal line:

Serratus magnus.
Trapezius (acromial).
Trapezius (inferior).

Muscle Action.—The first group of muscles carries the humerus forward nearly to the horizontal line. The trapezius and the serratus magnus contract to fix the scapula when the humerus approaches the horizontal line, the trapezius acting first, and the serratus magnus immediately afterward.

Test.—The patient, sitting or standing, can raise his arm to the horizontal line anteriorly.

EXTENSION OF THE HUMERUS.

The muscles that extend the humerus in the antero-posterior plane:

Pectoralis major (sternal fibers).
Latissimus dorsi.
Teres major.
Teres minor.
Infraspinatus.
Triceps (long head).
Subscapularis (?)

Hyperextension (carrying the arm back of the body):

Latissimus dorsi.
Teres major.
Teres minor.
Infraspinatus.
Deltoid (posterior fibers).

Muscle Action.—The first group of muscles brings the arm downward and forward, and the second group carries the arm back of the body. During this action, the scapula is fixed by the rhomboids, the lowest fibers of the trapezius, and the pectoralis minor.

Test.—In the test for extension of the humerus, the patient, standing or sitting with the arm raised above the head, brings it forward and down until it is in line with the body. In the test for hyperextension of the humerus, the patient, standing or sitting with the arm hanging at the side, moves it backward.

ABDUCTION OF THE HUMERUS.

Muscles that abduct the humerus in the latero-vertical plane:

To the horizontal line:
  Deltoid (middle fibers).
  Supraspinatus.
  Biceps.
Above the horizontal line:
  Serratus magnus.
  Trapezius (acromial and inferior fibers).

Muscle Action.—The first group of muscles raises the arm to the horizontal line, and the second group raises it from the side to above the horizontal line.

Test.—The patient, standing or sitting, can raise the arm sideways to the horizontal. In this test the palm should be directed downward to exclude the action of the biceps as much as possible.

ABDUCTION OF THE HUMERUS HORIZONTALLY.

Muscles that abduct the humerus horizontally:

Deltoid (middle fibers).
Deltoid (posterior fibers).
Latissimus dorsi.
Teres major.
Teres minor.
Infraspinatus.
Subscapularis.

Muscle Action.—This group carries the arm back at shoulder level.

Test.—The patient, in a sitting position with the arm in adduction, that is, crossed over to the shoulder of the opposite side, abducts the arm.

ADDUCTION OF THE HUMERUS IN THE LATERO-VERTICAL PLANE.

Adductor muscles of the humerus:

Pectoralis major (all).
Latissimus dorsi.
Teres major.
Teres minor.
Infraspinatus.
Subscapularis (?)
Deltoid (posterior third).

Muscle Action.—This group of muscles brings the arm to the side.

Test.—The patient, sitting or standing, draws the arm down to the side against resistance under the elbow. If the muscles are normal they can overcome considerable resistance.

ADDUCTION OF THE HUMERUS HORIZONTALLY.

The muscles that adduct the humerus horizontally:

Coracobrachialis.
Pectoralis major.
Deltoid (anterior half).

Muscle Action.—This group brings the arm toward the middle line of the body at shoulder level.

Test.—The patient, lying on his back with his arms stretched out on a level with the shoulders, the elbows straight and the palms up, can raise the arms upward until the palms meet.

ROTATION OF THE HUMERUS.

The muscles that rotate the humerus inward:

Pectoralis major.

ROTATION OF THE HUMERUS (Continued).

Deltoid (anterior fibers).
Teres major.
Latissimus dorsi.
Subscapularis.

The muscles that rotate the humerus outward:

Deltoid (posterior fibers).
Infraspinatus.
Teres minor.

Test.—The patient lies face down, with the arm stretched sideways at shoulder height, and the forearm and hand hanging over the edge of the table. The surgeon steadies the upper arm, and the patient attempts to raise the hand backward and upward against resistance on the ulnar side of the wrist, thus rotating the humerus inward. The test for outward rotation of the humerus is similar to that for inward rotation, except that the surgeon offers resistance against the radial side while the patient raises the hand forward and upward to rotate the humerus outward.

THE FOREARM.

FLEXION OF THE FOREARM.

Muscles that flex the forearm:

Biceps (flexor supinator).	Flexor muscles of the wrist.
Brachialis.	
Brachioradialis.	
Pronator teres (flexor pronator).	
Flexor carpi radialis.	
Flexor carpi ulnaris.	
Palmaris longus.	
Flexor digitorum sublimus.	Flexor muscles of the fingers.
Flexor digitorum profundus.	
Lumbricales.	
Interossei.	
Flexor brevis minimi digiti	
Extensor carpi radialis.	Extensor muscles of wrist and fingers in pronation.
Extensor carpi ulnaris.	
Extensor communis digitorum.	
Extensor longus pollicis.	

Muscle Action.—These muscles bend the forearm on the upper arm.

Test.—The patient, in a sitting position, can bend the elbow until the hand touches the shoulder. The test should be made both in pronation and supination. Care must be taken to note particularly the power of the biceps, as the prognosis of recovery of function in the arm is dependent upon the return of power in this muscle (Fig. 50).

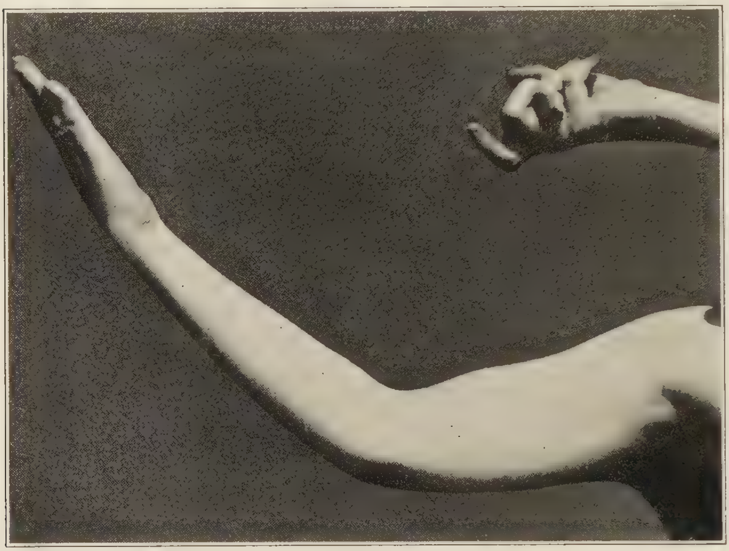

FIG. 50.—Test for flexion of the forearm.

EXTENSION OF THE ELBOW.

Muscles that extend the forearm:

Triceps.	Extensors of the wrist.
Anconeus.	
Extensor carpi radialis.	
Extensor communis digitorum.	
Extensor longus pollicis.	
Extensor carpi ulnaris.	
Extensor indicis proprius.	Extensors of fingers in supination.
Extensor minimi digiti.	
Lumbricales.	
Interossei.	

Muscle Action.—These muscles straighten the elbow.

Test.—The patient can extend his arm upward from a flexed position.

PRONATION OF THE FOREARM AND HAND.

Muscles that pronate the forearm and hand:

Pronator radii teres.
Pronator quadratus.
Flexor carpi radialis.

Muscle Action.—This group of muscles turns the palm downward.

Test.—The patient, sitting with his hand resting on his lap palm up, can turn the palm down against resistance offered by the examiner.

SUPINATION OF THE FOREARM AND HAND.

Muscles that supinate the forearm and hand:

Supinator radii brevis.
Biceps.
Brachioradialis (Cunningham).

Muscle Action.—This group of muscles turns the palm upward.

Test.—The patient, sitting with his hand resting on his lap palm down, can turn the palm upward against resistance offered by the examiner.

THE WRIST.

FLEXION OF THE WRIST.

The muscles that produce flexion:

Flexor carpi ulnaris.
Flexor carpi radialis.
Palmaris longus.
Long flexors of thumb and fingers.

Muscle Action.—This group draws the hand downward.

Test.—The patient sits with the forearm supported on the table, and the hand extending over the edge with the palm up. From this position he can flex the wrist against resistance offered by the surgeon.

EXTENSION OF THE WRIST.

Muscles that produce extension:

Extensor carpi radialis.
Extensor carpi ulnaris.
Extensors of thumb and fingers.

Muscle Action.—This group of muscles extends the wrist on the forearm.

Test.—The patient places his forearm on the table with the band extending over the edge palm down, and extends the wrist from this position.

ABDUCTION OF THE WRIST.

The muscles that produce abduction:

Flexor carpi radialis.
Extensor carpi radialis longior (Piersol).
Extensors of wrist and thumb.

Muscle Action.—This group of muscles turns the hand outward.

Test.—The patient rests his arm on a table, ulnar side down, with the hand extended over the edge of the table. He now tries to bring the wrist from the mid-line to the ulnar side.

ADDUCTION OF THE WRIST.

The muscles that adduct the wrist:

Flexor carpi ulnaris.
Extensor carpi ulnaris.

Muscle Action.—This group draws the wrist toward the radial side.

Test.—The same position is assumed as in the test for abduction, but the patient tries to draw the hand to the radial side.

THE FINGERS.

FLEXION OF THE FINGERS.

The muscles that produce flexion:

Flexor digitorum sublimis.
Flexor digitorum profundus.
Lumbricales.
Interossei (acting on the metacarpo-phalangeal articulations).
Flexor brevis minimi digiti.

Muscle Action.—This group of muscles enables the patient to make a fist.

EXTENSION OF THE FINGERS.

The muscles that produce extension:

Extensor digitorum communis.
Extensor indicis proprius.
Extensor minimi digiti.
Lumbricales.
Interossei (acting on the interphalangeal articulations).

Muscle Action.—This group of muscles straightens out the fingers.

ABDUCTION OF THE FINGERS.

The muscles that produce abduction:

Lumbricales.
Flexor brevis and opponens digiti quinti (from the medial side of the hand).

ABDUCTION OF THE FINGERS (Continued).

Dorsal interossei (from the middle line of middle finger).

Muscle Action.—This group abducts the fingers.

Test.—The patient, starting with fingers together, draws them away from each other.

ADDUCTION OF THE FINGERS.

The muscles that adduct the fingers:

Palmar interossei (to the middle line of the middle finger).

Muscle Action.—This group of muscles adducts the fingers.

Test.—The patient, starting with the fingers spread, brings them together.

THE THUMB.

FLEXION OF THE THUMB.

The muscles that flex the thumb:

Opponens pollicis (carpo-metacarpal joint).
Flexor brevis.
Adductor pollicis.
Abductor brevis (carpo-metacarpal and metacarpo-phalangeal joint).
Flexor pollicis longus (all joints).

Muscle Action.—This group flexes the thumb on the hand.

EXTENSION OF THE THUMB.

The muscles that extend the thumb:

Abductor pollicis (carpo-metacarpal joint).
Extensor pollicis brevis (carpo-metacarpal and metacarpo-phalangeal joint).
Extensor pollicis longus (all joints).

Muscle Action.—This group extends the thumb.

ABDUCTION OF THE THUMB.

The muscles that abduct the thumb:

Abductor pollicis brevis.
Extensors of the thumb.

Muscle Action.—This group of muscles draws the thumb away from the mid-line toward the radial side of the hand.

ADDUCTION OF THE THUMB.

The muscles that adduct the thumb:

Flexor pollicis brevis.
Opponens pollicis.
Interosseous (first dorsal).

Muscle Action.—These muscles draw the thumb toward the mid-line.

Circumduction of the Thumb.—Circumduction of the thumb combines the action of the muscles that produce flexion, extension, abduction, and adduction of the thumb.

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CHAPTER XIII.

THE SURGICAL TREATMENT OF PARALYSIS.

SURGICAL measures are used in paralytic conditions for two purposes: To correct a deformity which is interfering with normal joint function, and to improve the function of a paralyzed part.

Among the operative procedures used to correct deformity are: Manipulation, fasciotomy and tenotomy, tendon lengthening and shortening, and osteotomy. The selection of the operation applicable to a given case depends upon the degree of the deformity. If either a tenotomy or a manipulative measure is adaptable, there is no question as to the superiority of the stretching process. While a deformity may be corrected by tenotomy, the contracted muscle fibers are not lengthened in the process, and the muscle is left in a weakened condition. Manipulative and stretching methods, on the contrary, ensure the preservation of muscle strength. An occasional case is seen in which it is not possible to use the manipulative method, and tenotomy is necessary.

The operations that have for their object the improvement of function are: Tendon transplantation, tenodesis, astragalectomy, and certain stabilizing measures. Other methods, such as nerve transplantations, and stabilization by means of silk ligaments, have been devised for the purpose of improving function, but the results of these procedures have not been gratifying. Direct transplantation of a live nerve into the distal end of a severed paralyzed one has thus far proved of no value clinically. Also, the grafting of a live nerve to a paralyzed one has not been satisfactory. The use of silk ligaments is based upon the theory that the silk becomes surrounded by scar tissue and forms ligaments which maintain the proper anatomical relations. It is in few cases that the expected result is obtained; either the silk stretches, or, what more often happens, it acts as a foreign body and causes irritation, so that its removal is necessary. In other cases, the silk may hold the foot firmly in position, but it is impossible for the artificial ligament to adapt itself to changes of growth, and deformity results.

Indications.—When deformity is progressing in spite of conservative treatment, or when function is impaired because of muscle unbalance, operative interference is indicated. If possible, such measures should not be used until two years have elapsed from the onset of the disease, for during this period there is always the possibility that the muscle power will improve under conservative treatment. The exceptional case, in which operation is indicated before the two-year convalescent period has elapsed, is one of increasing deformity.

As to the best age for operation, stabilizations and other radical operative procedures that have for their purpose the improvement of muscle and joint balance are more efficient when the child is at least seven, or better, twelve years of age.

Preparation for Operation.—The preparation for operation should be carried out according to the following routine. The child should enter the hospital two days before operation. Sodium bicarbonate (gr. x. t. i. d.) with small amounts of glucose should be administered to control acidosis, especially in frightened children. The blood and urine should be made alkaline.

Two days before operation, the area including one joint above and one joint below the operative field should be shaved, scrubbed with soap and water, and a dry sterile dressing applied. On the second day, the operative area is painted with a weak solution of iodine and exposed to the air for fifteen minutes. A dry sterile dressing is then applied, which remains on until the morning of the operation.

Orange juice is given several hours before operation, and an enema is also advisable.

Ether is used for an anesthetic.

Asepsis.—Meticulous asepsis in all bone and joint surgery is most important. The operative area must be carefully prepared and properly draped. The skin should not be handled by the gloved fingers, but should be clamped off with towels after the incision has been made. A tourniquet is frequently used to control hemostasis.

Sutures.—Chromic gut is used for ligatures, as it is strong and absorbable. When tension is needed for only a few days, catgut is the best material to use. Catgut is used largely for skin sutures when plaster bandages are applied. Pagenstacker sutures may be used when non-absorbable material is required. The use of large amounts of suture material should be avoided, and knots should be placed so as to minimize mechanical irritation.

After-care.—The after-care is vitally important to the success of all operations for paralytic conditions. Routine after-treatment is not sufficient; frequently the patient requires attention throughout years of convalescence, and only one who is thoroughly trained for such work is capable of supervising the treatment. The application of the plaster bandage is a study in itself, for it must not only hold the part in the position required, but it must be comfortable, efficient, light, and durable. Massage, exercise, and instruction in balance are salient factors in the after-care of paralytic cases.

Results of Operation. –The results which may be expected from the use of surgical procedures in paralytic cases are discussed under the section covering each deformity. In general, we may say that deformity can be corrected, and that in the majority of cases, muscle weakness and instability can be lessened. The success of any surgical measure depends upon the execution of the operative details with meticulous care, and upon the thoroughness with which the after-care is carried out.

Dangers of Operation.—Provided the operation is carefully performed and strict asepsis maintained, there is little danger. Acetonuria is common in children and occasionally becomes serious. It should be overcome by obviating fright and administering sodium bicarbonate and glucose for several days before operation.

MANIPULATION AND STRETCHING.

It is possible, by manual stretching and manipulation, to correct many cases of tissue contracture, and to assist in restoring to normal some cases of slight structural deformity. When the deformity is a mild bony one, the stretching of the joint capsule and contracted tissue will restore the joints to proper relation, and if the part is maintained in corrected position for sufficient time, the smaller bones will resume their normal relations. Manipulation usually can be carried out by the manual method. When the patient has been anesthetized, the limb is forced slowly with increasing pressure into overcorrection, the degree depending upon the severity of the deformity and the length of time it has existed. Once correction has been obtained, a plaster bandage is applied to maintain the limb in position. If the circulation does not return to normal shortly after manipulation, it is often necessary to lessen tension by bivalving the plaster. Several attempts should be made to correct a contracture by manipulation before resorting to surgical measures.

The dangers of a manipulation are slight provided the process is performed with care. In severe contractures, it is well to secure overcorrection slowly by repeating the manipulative process; in such cases the vessels, nerves, and ligaments are contracted, and a too forcible manipulation may cause such trauma that gangrene would result.

Manipulation and Stretching of the Foot.—A description of the methods of manipulation used in the correction of a talipes varus and a talipes equinus will serve to illustrate the procedure carried out to overcome any contracture of the foot.

In varus deformity the foot is forced slowly, with increasing pressure, into the position of valgus (Fig. 51). In very resistant cases, a Thomas wrench may be used to supply the leverage and force necessary to obtain overcorrection. A plaster bandage, extending from the toes to the knee, is then applied to hold the foot in the position of valgus.

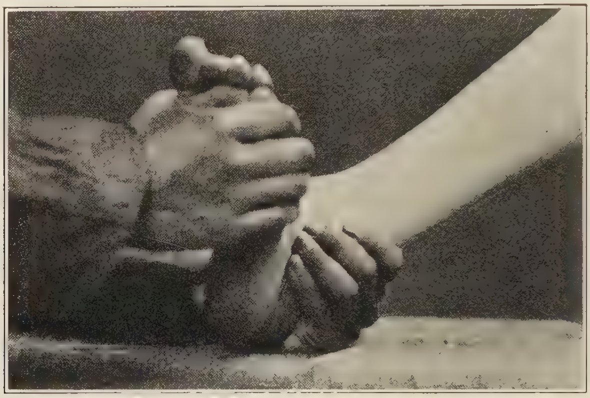

FIG. 51.—Talipes varus. Manipulation of the foot into valgus.

In talipes equinus the leg is extended and held by an assistant. The foot is forcibly pushed into dorsal flexion by slow, steady movements. If necessary a Thomas wrench is used. When the foot is in 15 degrees dorsal flexion, a plaster bandage, extending from the toes to just below the knee, is applied. The circulation of the toes must be watched carefully during manipulation and following the application of the bandage.

The manipulative procedure, repeated if necessary, is so successful in overcoming practically any degree of Achilles contraction that, except in rare cases, the writer refrains from performing open operations on the Achilles tendon. Following a stretching process, return of power in the gastrocnemius is nearly complete, whereas the function and strength in this muscle never return to normal after a tenotomy of the tendo Achillis.

After a manipulation the foot is maintained in a plaster bandage for six to eight weeks. Weight-bearing is encouraged as soon as tenderness has subsided to prevent early atrophy from disuse and to favor muscle hypertrophy. When the plaster has been removed, massage, and active and passive movements are indicated. It is well for the patient to wear some form of apparatus for a short time to prevent the return of contracture.

The same principles of manipulation and after-care are applied in the correction of other deformities of the foot. In associated deformities, such as talipes calcaneovalgus, the foot is first manipulated into equinus and then forced into varus.

In extreme structural deformity, manipulations are used to overcome tissue contracture preliminary to correction by open methods.

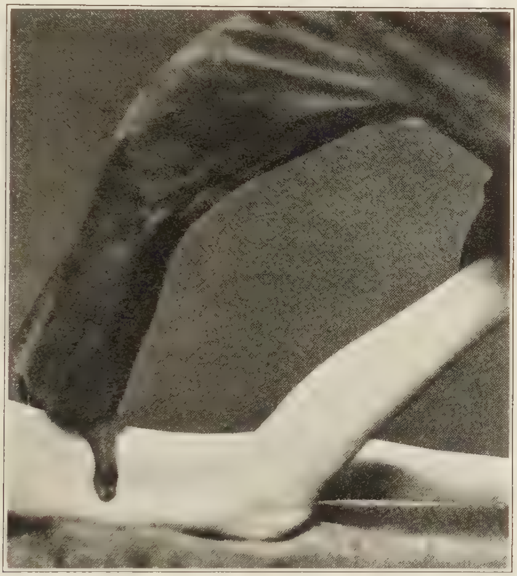

FIG. 52.—Contracted knee. Manipulation of the knee into extension.

Manipulation and Stretching of the Knee.—In mild contracture of the hamstring tendons, manipulation is performed with the patient lying on his back. The operator places one hand above the knee to secure leverage, and with the other hand placed below the knee, he stretches the hamstring tendons by slow, increasing pressure. When the leg is straight, a plaster bandage is applied from the toes to the groin. During application of the plaster, the leg is held in extension by an assistant, who grasps the patient's heel in his hand and exerts pressure on the femur by resting one foot in a 4-inch bandage, which has been looped around the knee.

In old-standing contractures in which the vessels, nerves, and ligaments in the popliteal space are involved, or in flexion deformity of the knee associated with subluxation, manipulation is performed more safely with the patient face downward. An assistant holds the well leg and the pelvis firmly on the table. The operator, with one hand placed above the knee and the other hand placed well below the knee, applies a gradual pressure (Fig. 52). This procedure is more frequently used than the one for simple contracture, as flexion deformity of the knee is often accompanied by some subluxation.

In severe contractures, it may be necessary to divide the manipulative process into several stages, gaining slight correction each time. There should be an interval of several weeks between each stage.

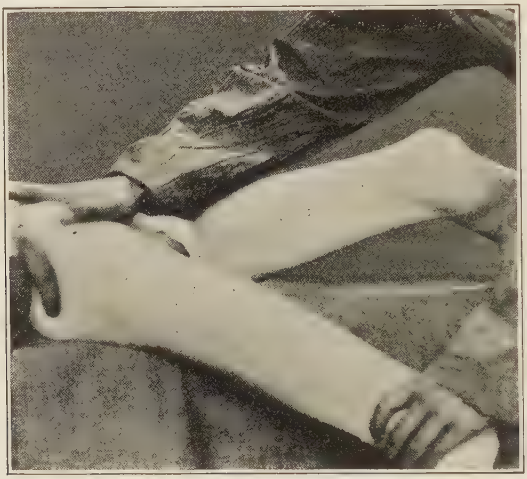

Fig. 53.—Contracted hip. Manipulation of the hip into hyperextension.

Following manipulation, a plaster bandage is applied from the toes to the groin. The toes should be watched carefully for the return of circulation; if this is not established shortly, the plaster bandage should be split anteriorly to relieve tension. In simple cases the knee is kept in plaster for six to eight weeks. A caliper brace should be worn for several months after the correction of deformity to prevent recontracture. Local physiotherapy should be started as soon as the plaster bandage is removed.

Manipulation and Stretching of the Hip.—Flexion Deformity.— The patient lies on his back with the contracted thigh hanging off the table. Assistants hold the opposite leg in flexion and grip the pelvis firmly. The operator, using the knee and thigh for leverage, slowly and with rhythmic movement, forces the leg into hyperextension and abduction (Fig. 53). Care should be taken to obtain a good overcorrected position. If this is not possible by one manipulation, the procedure may be repeated. It is often difficult to correct flexion deformity of the hip.

A plaster spica is applied from the breast-line to below the knee, with the leg held in 15 degrees hyperextension and 5 to 10 degrees abduction. If deformities of the knee or ankle have required manipulation at the same time, the bandage may be extended to maintain these parts in overcorrected position. The plaster spica is worn for six weeks to two months. Massage and exercises are then begun, and apparatus is worn if necessary.

Abduction Contracture.—In abduction contractures, forcible manipulation of the leg into hyperextension and adduction should be done as a supplementary measure to a fasciotomy of the tensor vaginæ femoris. Manipulative treatment alone seldom is sufficient to effect correction of abduction deformity.

Manipulation of the Shoulder into Abduction.—An assistant holds the scapula to the side, while the operator forcibly manipulates the arm into abduction, backward extension, and external rotation. With the arm in this position, a plaster spica is applied around the chest and shoulder, and down the arm as far as the finger tips. The bandage remains on for at least six weeks. If, upon its removal, the deformity has not been fully corrected, the manipulation may be repeated several times before operative interference is considered.

When the plaster bandage has been discarded, an abduction arm splint should be worn to eliminate the effect of gravity on the paralytic shoulder and to prevent a tendency to recontracture.

FASCIOTOMY.

Fasciotomy of the Plantar Fascia for Cavus Deformity.—This operation when combined with a manipulation serves to correct mild cavus deformity.

Operative Technique.—The assistant forces the foot into dorsiflexion. A tenotome is inserted in a slanting direction about half way between the internal malleolus and the sole of the foot (Fig. 54). The tenotome, guided by the operator's finger, is passed subcutaneously to a point directly over the plantar fascia. The cutting edge of the tenotome is turned toward the fascia, and all tight bands are divided. The foot is then forcibly manipulated into dorsiflexion.

A plaster bandage is applied from the toes to the knee with the foot in dorsiflexion. This is worn six weeks, weight-bearing being permitted after the first week. Local therapy, and stretching to prevent recontracture, are advisable upon the removal of the plaster.

The Stripping of the Os Calcis (Steindler[1]).—This is a more radical procedure than a fasciotomy, but it offers a better chance of securing correction of severe tissue contracture. It also may be used as a preliminary measure to an osteotomy or a wedge-shaped resection in cases of structural cavus deformity. The stripping is of no value in severe paralysis, especially if the calf muscles are involved and calcaneus deformity is present. Stabilizing operations are required in such cases.

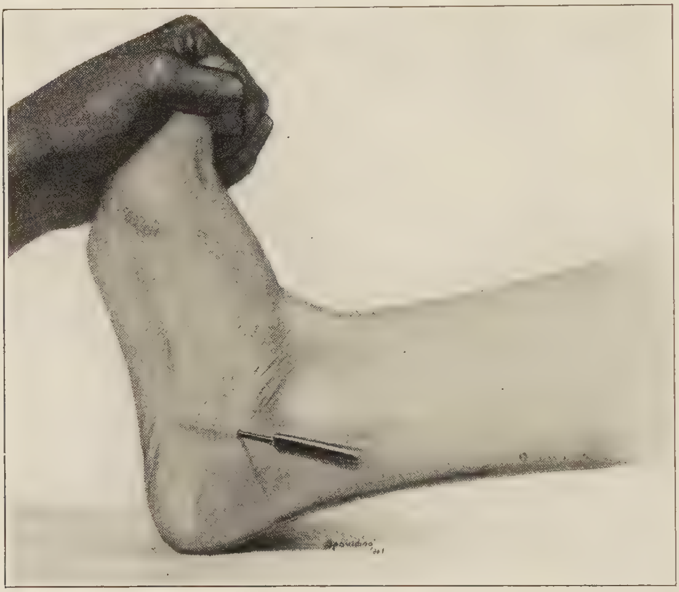

FIG. 54.—*Fasciotomy for cavus deformity.* Insertion of the tenotome half way between the internal malleolus and the sole of the foot.

The stripping of the os calcis is based upon an anatomical condition that is favorable to such an operation—that is, the muscular attachments of the small muscles of the sole of the foot are grouped about a circumscribed point at the posterior process of the os calcis.

Operative Technique.—"A horizontal incision is made over the inner aspect of the os calcis forward, reaching to a point 1½ inches in front of the inner tubercle of this bone. When the skin and fat are divided, the lower or plantar surface of the plantar fascia is separated from the layer of fat and freed throughout its entire width. The fascia is then incised crosswise close to the point where it blends into the lower surface of the os calcis. The muscles covered by the plantar fasciæ are from medially to laterally, the abductor of the big toe, the short flexors of the toes, and the abductor of the fifth toe. These muscles are now stripped off the periosteum of the os calcis with a blunt instrument and it is necessary to extend the stripping forward to the calcaneo-cuboid junction in order to reach and to strip off the ligamentum plantare longum which extends between the os calcis and the cuboid. This ligament is often contracted, and if so, produces a cavity of the foot at the outer border.

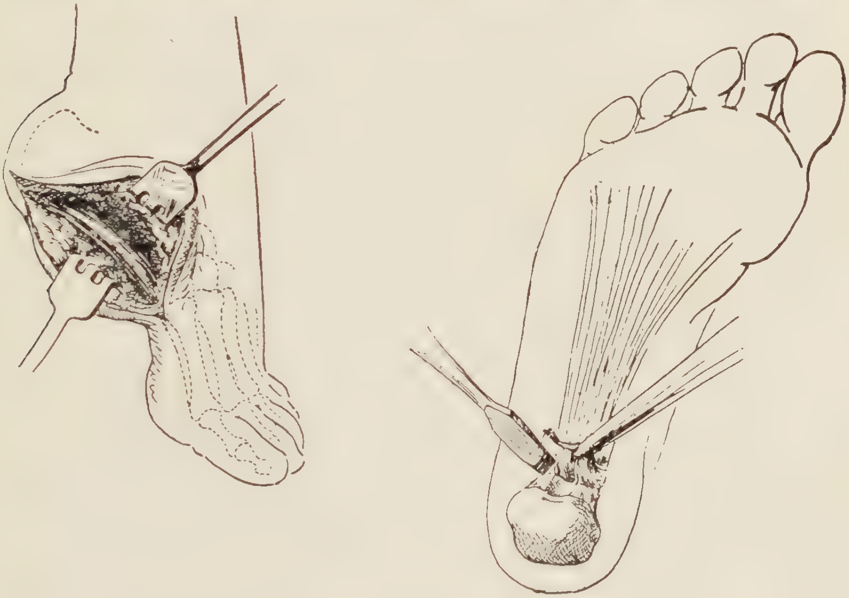

Fig. 55. Fig. 56.

Figs. 55 and 56.—Stripping of the os calcis for cavus deformity. (Steindler.)

By keeping close to the bone in stripping, one is at a safe distance from the plantar vessels and nerves. When the stripping has been completed, the correction of the foot is accomplished by wrenching and in severer cases by such bone operations as it may be necessary to add according to the degree of the skeletal deformities (Figs. 55 and 56).

"One should avoid taking along any cortical bone from the os calcis when the stripping is done. Unless this point is observed a good deal of bone formation may occur at the plantar surface of the foot. This complication need not occur, however, if one is careful to use a blunt instrument or separate the muscles close to the periosteum with a knife instead of scraping sharply with a keen-edged chisel.

"For the purpose of correcting the cavus deformity by stripping and subsequent wrenching, it is quite necessary to have the advantage of the leverage of a tight and solid tendo Achillis. Therefore, the tenotomy of this tendon should never be done together with this operation. If it is to be done, it should either precede or follow the stripping operation in intervals long enough for the purpose. But in discussion on the indication for lengthening of the tendo Achillis, it has been remarked that in cavus deformity of the foot the shortening of this tendon in general is not nearly as considerable as generally assumed or as it might possibly appear upon first glance. The deviation of the foot downward occurs largely in the mid-tarsal joint and not in the ankle-joint so that in most instances the tendo Achillis is not considerably shortened and, therefore, need not be lengthened. An additional procedure which completes the correction of claw-foot is the implantation of the extensor tendon of the toes to the metatarsal bones for the purpose of lifting the transverse arch of the foot (R. Jones). In paralytic cases such procedures may be necessary for the restoration of the balance. Otherwise, however, a complete correction by stripping and other corrective operations usually suffices in the mild and moderate cases. The severe hollow foot which follows extensive paralysis does not hold correction well, but this is because of the disturbance of the muscle balance which always persists after anatomical correction has been completed. . . It is far more rational to relegate these cases to the arthrodesing and other stabilizing operations of the foot."

Steindler has operated 91 cases of paralytic cavus by the method of stripping the os calcis. The results in 90 per cent of the cases were good; the plantar fascia showed no tendency to contract. The 10 per cent failures were due to error in the selection of cases for the stripping procedure.

Case V.—A. E., aged five years. (Courtesy of Dr. Steindler.)

Complaint: Foot turns in. Condition getting worse.

Duration: Four years.

Cause: Infantile paralysis.

Physical Examination: Left foot showed marked cavovarus deformity (Fig. 57).

Treatment: *September 4, 1920.* A stripping of the os calcis and an osteotomy of the neck of the astragalus were performed, and a good correction obtained (Fig. 58). The patient did not return for treatment regularly, and deformity recurred (Fig. 59).

June 10, 1924. Another stripping and wrenching operation was done, and a fair correction secured.

July, 1925. The foot was fairly well corrected. The patient was still wearing an Orr brace, varus strap, outer wedge, and anterior heels on the shoes.

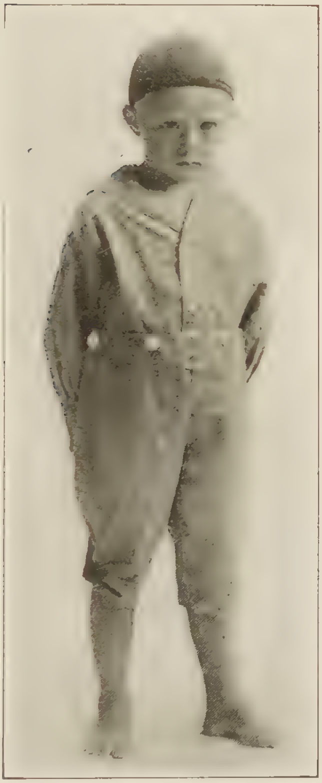

Fig. 57.—Before first stripping operation.

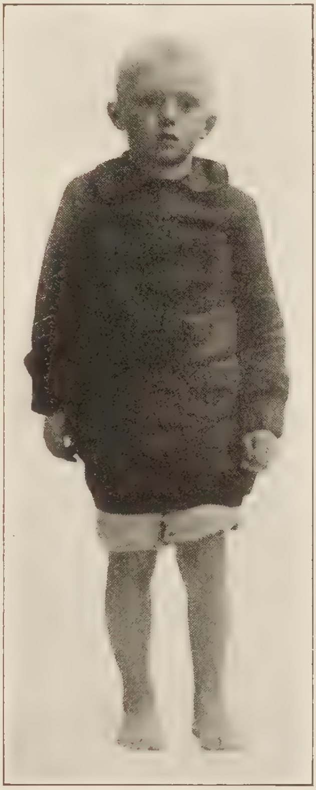

Fig. 58.—Correction, four months after first stripping operation.

Figs. 57 to 60.—Case V. A. E. *Cavovarus deformity of the left foot.* (Courtesy of Dr. Steindler.)

July 17, 1925. The foot was wrenched again, and a good correction obtained.

Result: February, 1926. At last observation, slight cavoequinus was still present, but it was well corrected on weight-bearing. The patient was wearing anterior heels and outer wedges on the left shoe (Fig. 60).

Fasciotomy for Flexion Deformity of the Hip.—The principle of this operation lies in lowering the origin of the tensor vaginæ femoris.

The procedure may be used in the more resistant and severe contractures of the hip, in which manipulation has failed. Precaution must be taken in performing this operation to avoid a too forcible manipulation, which would affect the abdominal viscera as well as the femoral artery and the anterior crural nerve. Ileus frequently occurs after this operation.

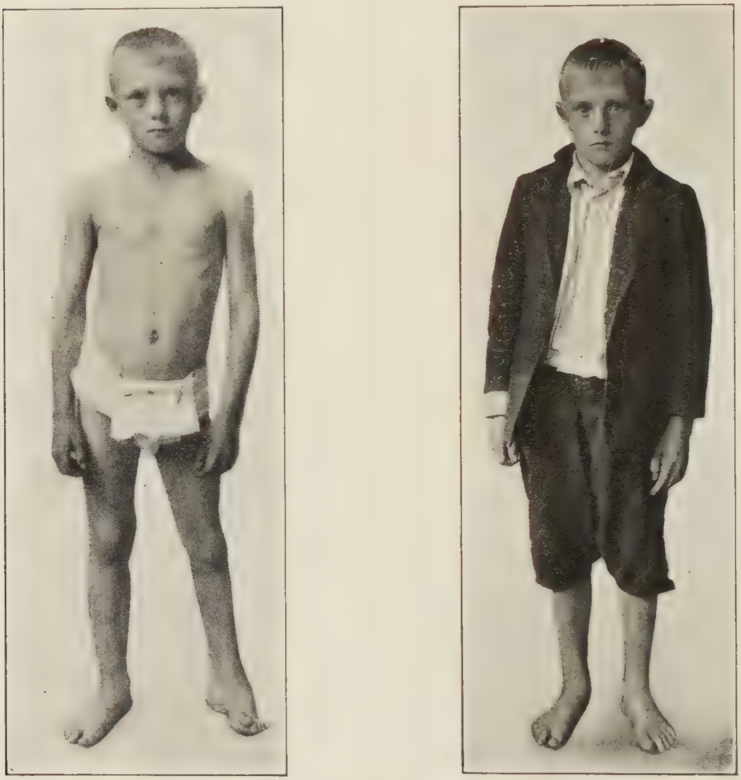

FIG. 59.—Showing recurrence of the deformity, due to parent's neglect to carry out the after-treatment.

FIG. 60.—Correction, one year, eight months after second stripping operation.

FIGS. 57 to 60.—Case V. A. E. *Cavovarus deformity of the left foot.* (Courtesy of Dr. Steindler.)

Operative Technique (Soutter[2]).—A longitudinal incision, about 3 inches in length, is made along the anterior edge of the tensor vaginæ femoris, midway between the anterior-superior spine and the trochanter. The fascia is exposed and incised at right angles to the skin incision, all its fibers being cut transversely from the anterior-superior spine nearly to the great trochanter. The skin incision is retracted to expose the anterior-superior spine. By means of an osteotome, the attached muscles and fascia are stripped subperiosteally from the anterior-superior spine. The hip is hyperextended. If the tension is too great, the soft tissues and periosteum are pushed down from part of the pelvis below the anterior-superior spine. The crest of the ilium is smoothed off, and the wound closed.

The form of after-treatment depends upon the amount of correction obtained at operation. If full extension is gained, a double hip spica is applied at once, and the patient placed in bed on a frame. If complete correction is not secured, the patient may be placed in bed on a frame, and extension obtained by means of a Thomas splint and an extension pull. It is also possible to ensure complete extension by applying a hip spica in two sections, one on the leg and one on the trunk. After the hip has been stretched, the sections may be united.

Results.—Steindler[1] used this operation in 69 cases of flexion contracture following infantile paralysis. Examination of 63 of these cases, one year or more after operation, showed that 88 per cent had complete extension both in abduction and adduction.

THE SURGERY OF TENDONS.

Tenotomy of the Achilles Tendon.—Since the year 1838, when Stromeyer[3] elaborated the method of a subcutaneous tenotomy of the Achilles tendon, the operation has been used extensively. In the early application of this method its value was never questioned, in spite of the fact that the results were not always favorable. At the present time, stretching procedures have replaced tenotomy to a great extent and are much to be preferred, since they allow the muscle strength to be preserved.

Open operation may be necessary when lengthening of the contracted tendons cannot be effected by stretching. The operation should be done subcutaneously, and some of the muscle fibers should be torn, not cut, in order to prevent non-union. The writer believes that the non-union often resulting in adult cases is due to the severing of all continuity by a transverse section of the sheath and tendon.

Operative Technique.—The best method of procedure is to divide the tendon obliquely. The tenotome is introduced about ½ inch above the tip of the os calcis, and passed under the skin and fascia to a point about 1½ inches above the insertion of the tendon into the os calcis. The tenotome is then directed toward the tendon, and this is cut in a slanting direction. At the time of cutting, the foot is forced steadily into flexion, to enable the tearing of as many fibers as possible.

After the tendon has been divided, the foot is dorsally flexed to about 15 degrees above a right angle, and a plaster bandage applied from the toes to above the knee. Weight-bearing may begin within a few days. At the end of the third week, the plaster bandage is removed and a lighter one applied. This is worn for about three weeks. The customary after-treatment, including massage and exercises, is advisable upon the removal of the plaster bandage.

In children, the tendon unites in about three weeks, and in adults in about six weeks.

Elongation of the Achilles Tendon.—This operation is used occasionally in adult cases. It should not be used until repeated stretchings have been tried, because weakness of the gastrocnemius may follow operative elongation of the tendon. The procedure is performed with little surgical risk.

Operative Technique.—An incision, 3 to 4 inches in length, is made through the skin and fascia along the inner side of the Achilles tendon, extending upward from the side of the os calcis and running parallel to the tendon. The skin and fascia are retracted. An incision is then made in the sheath of the tendo Achillis, and the sheath retracted. A longitudinal incision is made in the mid-line of the tendon, and transverse cuts are made at each end of this incision, one running inward to the longitudinal incision and the other outward from it. Lengthening is brought about by sliding the halves of the tendon along on each other (Fig. 61). The foot is then manipulated into dorsal flexion to overcome any contraction of the posterior ligaments. A Thomas wrench is helpful in reducing contractures of long-standing. The opposing halves of the tendon are united with chromic catgut or silk. The tendon sheath is closed with continuous fine catgut, and the skin is sutured with continuous catgut. A plaster bandage is applied from the toes to just above the knee, with the foot in 10 to 15 degrees dorsal flexion.

The period of convalescence is longer in a case of tendon lengthening than in a simple tenotomy. The plaster bandage is worn for eight weeks. Weight-bearing should be encouraged as early as possible, while the foot is still in plaster. Upon the removal of the plaster, the foot should be protected by a flannel bandage. Gentle massage and exercise are of advantage in reëstablishing circulation and function.

Case VI.—E. T., aged twenty-six years.

Complaint: Deformity and pain in right foot.

Duration: Twenty-five years.

Cause: Infantile paralysis.

This patient had an attack of infantile paralysis when fourteen months old. The Achilles tendon had been manually stretched when he was five years old. The child wore a brace up to the age of fourteen years, and his activity was only slightly restricted. Two years before the writer saw the patient, the foot began to give trouble, and the condition was gradually growing worse.

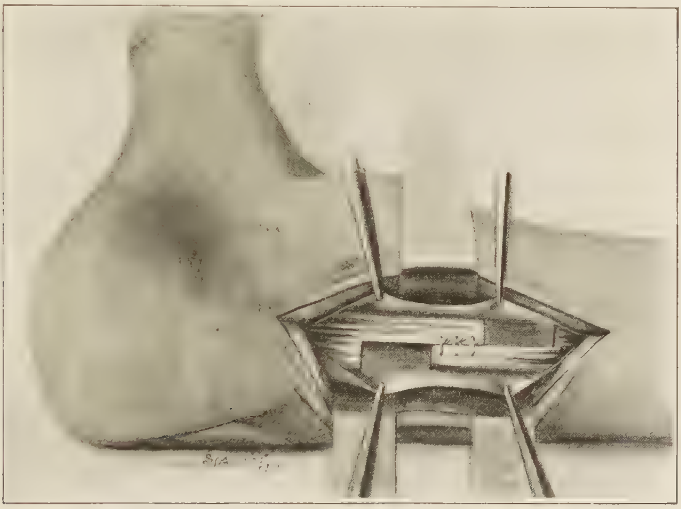

Fig. 61.—*Elongation of the Achilles tendon for talipes equinus.* Showing the sliding of the halves of the tendon along on each other to secure lengthening.

Physical Examination: November 17, 1925. The right foot was in the position of equinus, and the toes were considerably contracted. The foot could be brought only to within 15 degrees of a right angle. The right foot was ½ inch shorter than the left. There was good power in the peronei and in the dorsiflexors, especially in the extensor proprius hallucis. There was slight power in the tibialis anticus. Callous formations had developed under the anterior arches.

The right leg was considerably atrophied, particularly in its lower part, and was 1¼ inches shorter than the left.

Treatment: November 30, 1925. A tendon elongation was performed. An incision was made on the inner side of the Achilles tendon and carried down to the tendon sheath. The sheath was then incised. The Achilles tendon was split, and the halves allowed to slide by each other for about 1¼ inches. The tendon was sutured with chromic catgut No. 2, and the tendon sheath with continuous fine catgut. The wound was closed. The toes were manipulated into plantar flexion. A plaster bandage was applied to hold the foot above a right angle, and was extended down over the toes to hold them in plantar flexion.

December 22, 1925. A shoe with an outer upright, inner T-strap, and a right angle stop-joint was advised. In addition, a cork wedge was to be worn to compensate for the shortening.

Result: May, 1926, six months after operation, the foot could be flexed 5 to 10 degrees above a right angle. The callosities under the metatarsal heads had disappeared. The toes were less contracted and had more voluntary power of flexion (Fig. 62).

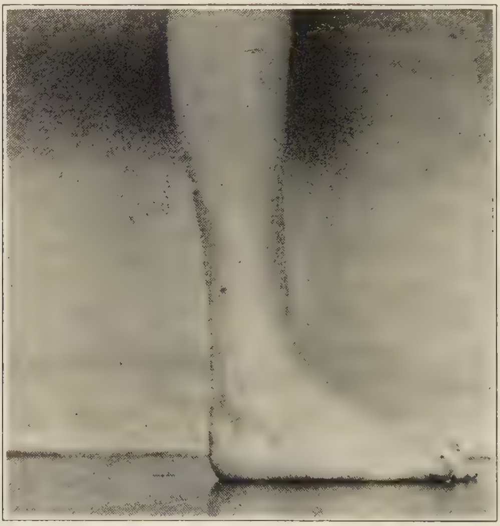

Fig. 62.—Case VI. E. T. *Talipes equinus.* Lateral view six months after tendon elongation.

Shortening of the Achilles Tendon.—Cases that require this operation are not common, for seldom is the Achilles tendon so overstretched as to warrant its use. The operation may be required if the tendon is severely stretched as the result of having been tenotomized before a forefoot deformity was corrected.

Operative Technique.—The foot is brought to a right angle in order to judge the amount the tendon must be shortened. An incision 3 inches long is then made through the skin and fascia along the inner side of the Achilles tendon, beginning about ½ inch above its attachment to the os calcis, and running parallel to the tendon. The skin edges are clamped off in the usual manner. The sheath of the Achilles tendon is incised and retracted. Two oblique incisions are made in the tendon, and the section between them, corresponding in size to the amount the tendon is to be shortened, is excised (Fig. 63). The severed ends of the tendon are then approximated with interrupted sutures.

The tendon sheath is closed with fine catgut, and the skin with continuous catgut No. 1.

The foot is brought into 5 degrees plantar flexion, and a plaster bandage applied from the toes to the knee. The plaster remains on for six to eight weeks. Walking on the plaster bandage is allowed when the tenderness has subsided. Upon the removal of the plaster, massage and exercises are advisable.

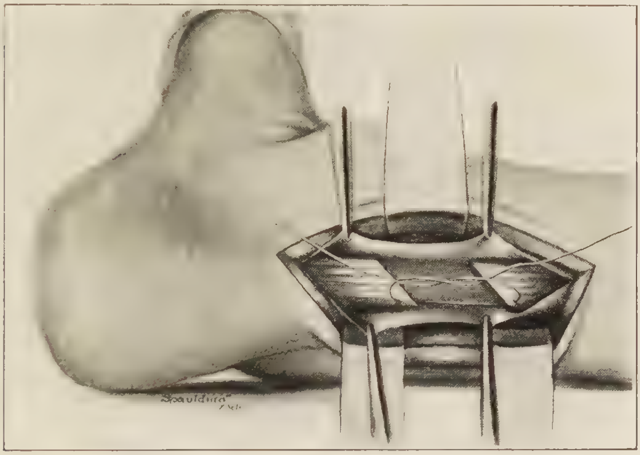

Fig. 63.—*Shortening of the Achilles tendon for talipes calcaneus.* The section of the tendon has been removed, and the ends are being approximated.

Case VII.—A. P., aged ten years.

Complaint: Deformity of the right foot.

Duration: Eight years.

Cause: Infantile paralysis.

After the initial attack, the right foot was in the position of talipes equinus, and the Achilles tendon, the plantar fascia, the forefoot, and the toes were contracted. Unfortunately, before the forefoot deformity had been corrected, a tenotomy of the Achilles was performed by another surgeon. As a result, the foot developed a calcaneovarus deformity.

Treatment: July 1, 1921. The foot was manipulated into valgus.

August 18, 1921. An arthrodesis of the calcaneo-astragaloid joint was done, and the Achilles tendon was shortened.

Result: November 16, 1925. The foot was stable and in good position. There was a slight tendency to cavus deformity of the forefoot (Fig. 64).

Division of the Hamstring Tendons.—Resistant deformity of the knee which cannot be overcome by stretching may be corrected by division of the hamstring tendons. The open method of operation is preferable to a subcutaneous procedure.

Operative Technique.—With the patient lying on his back, the leg is gently forced into extension. The patient is then turned on his face. An assistant extends the lower leg and holds it as close to the table as possible, making the hamstring tendons stand out prominently. An incision 2½ inches long is made over the inner hamstring group, and the skin and fat are retracted. The tendon is grasped with two snaps ¾ inch apart, and the section between the snaps is excised. The same procedure is then carried out on the outer hamstring group (Fig. 65). Any constricting fascia which appears to be holding the knee in a flexed position is divided. The wounds are closed with continuous catgut.

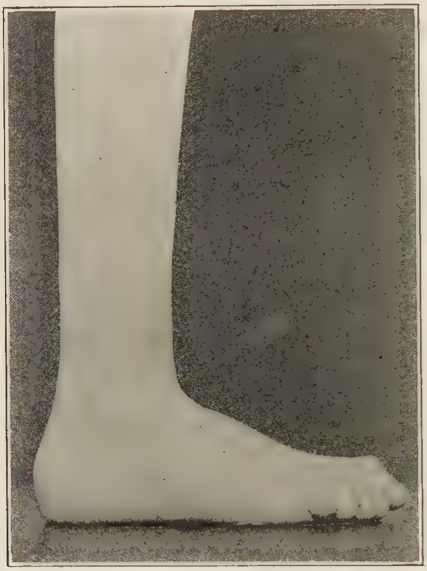

Fig. 64.—Case VII. A. P. *Talipes calcaneovarus.* Lateral view four years after shortening of the Achilles tendon.

The leg is extended, care being taken not to use too much force. A plaster bandage is applied from the toes to the groin. The circulation should be watched carefully.

The plaster bandage is worn for eight weeks. If extension for a longer period is necessary, a caliper brace may be worn. Massage, gentle manipulations, and exercise are advisable upon the removal of the bandage.

Division of the Sterno-cleido-mastoid.—Contractures in the cervical region, which cannot be overcome by traction or manipulation, may be corrected by division of the fascia and the sterno-cleido-mastoid tendon. The tenotomy is performed most satisfactorily by the open method. Some operators still use the subcutaneous procedure, but it involves more danger.

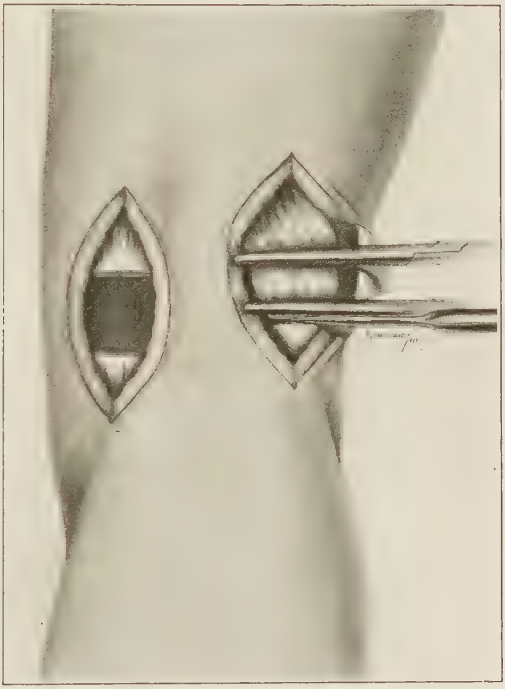

Fig. 65.—*Division of the hamstrings for flexion contracture of the knee.* The section has been removed from the inner hamstring, and the section to be removed from the outer hamstring is grasped with snaps.

Operative Technique.—An incision about 1 inch in length is made over the upper part of the clavicle, at the attachment of the sterno-cleido-mastoid. After the fascia and the platysma myoides have been divided, the sterno-cleido-mastoid muscle is severed as near its insertion as possible. The deformity is overcorrected very cautiously by manipulation, and the wound closed with subcutaneous catgut.

The head is held in an overcorrected position for six to eight weeks by means of a plaster bandage. Some form of retentive apparatus, or a plaster helmet, is then worn for several months to prevent recontracture. Massage and exercises are carried out after the removal of the first plaster bandage.

Tendon Transplantations.—A tendon transplantation aims to replace a paralyzed muscle by a normal muscle that is physiologically fit to act as a substitute. The use of transplantations in the treatment of infantile paralysis dates back to the year 1882, when Nicoladoni[4] transplanted one of the peroneal tendons into the tendo Achillis to correct a case of talipes calcaneus. The failure of this attempt and other efforts discouraged further experimentation along this line for several years. In the nineties, interest in tendon transplantations was renewed, and several good results led to an appreciation of its possibilities. Surgeons in Germany, Italy, England, and America experimented with this new method, and many advances and modifications were made in the technique of the operation. Codivilla[5] contributed much to the development of the procedure, and it is upon his work that the modern method is based.

The value of the operation and the permanence of its results have been subjects much discussed. When the first good results were obtained, many surgeons became enthusiastic advocates of the method and recommended its extensive use. In many instances, the physiological principles underlying transplantation procedures tended to be overlooked. Gradually, as the factors important to the success of these operations began to be appreciated, surgeons selected cases for transplantations more carefully and defined the limitation of the operation more precisely.

In the opinion of the writer, the results of tendon transplantations do not measure up to what is generally expected of them. An actual increase in function is seldom gained by the use of any of these procedures. Tendon transplantations are used most successfully as a means of improving the balance in single muscle group paralysis, and as supplementary measures to stabilizing operations in more extensive paralysis.

The only transplantations that give uniformly good results are: Transplantation of the anterior tibial tendon into the cuboid for talipes varus; transplantation of the flexor group in quadriceps paralysis as an aid to stability; and transplantations in paralysis of the wrist and fingers. Transference of the peronei into the scaphoid is of value in valgus deformity when used in combination with a stabilizing operation, and transplantation of the peronei into the Achilles tendon is satisfactory as a supplementary measure to an astragalectomy.

Provided deformity can be held in check by conservative measures, tendon transplantations should not be performed until the residual damage and distribution of the paralysis can be definitely estimated. This is usually a matter of two years. If the patient is too young to coöperate in the after-treatment, it is often advisable to postpone intervention longer. Usually, the child is not sufficiently developed mentally before the age of eight or nine years, to concentrate and assist in the reëducation of muscles after transplantation.

When, in spite of conservative treatment, deformity develops before the two-year period has elapsed and before the child is eight years of age, tendon transplantations may be indicated.

Although each case in which tendon transplantation is considered presents a different anatomical problem, which must be carefully worked out, there are certain principles applicable to all these operations.

Correction of Deformity.—Deformity must be corrected before transplantations are performed. Slight deformity may be corrected by simple manipulation at the time the transplantation is done. If the deformity is of moderate or severe degree, it must be well overcorrected before transplantation, either by manipulation or open operation. The period before transplantation, during which the foot is maintained in overcorrection, varies from one to six months, depending upon the severity of the deformity and the mechanical problem involved.

Selection of the Tendon for Transplantation.—The muscle selected for transplantation must be within the paralyzed area, and it must have the qualifications that are required for a successful substitution of power.

The muscle must be a healthy one. Its strength should be equal or approximately equal to the power of the muscle that it is to replace. This requirement is most important in weight-bearing limbs, in which stability is essential to the function of the joints. Since paralysis of the lower extremities involves largely the strong anterior and posterior groups, and there are no muscles that have sufficient strength to replace them, the use of transplantations in this region is limited. In the upper extremities where stability enters little into the problem, the necessity for equality of power between the paralyzed muscle and the tendon to be substituted is not so arbitrary.

The function of the tendon to be transplanted should approximate that of the paralyzed muscle. As a rule, well coördinated function does not result from transplantation of antagonistic muscle groups. Care should be taken to distinguish between muscles that have actual opposing function and those that act as stabilizers. For example, the flexors of the wrist exert a stabilizing power when the fingers are extended, and since they are not true antagonists, they may be transposed successfully to act for the paralyzed extensors.

The Preservation of the Muscle Unit.—The tendon should always be transferred *in toto*. The splitting of the tendon, for the purpose of allowing one-half of it to carry on its original function, is an unsatisfactory procedure.

The Gliding Mechanism.—It is very important to provide a gliding apparatus for the transplanted tendon. The writer removes the tendon from its sheath and places it beneath fat and fascia. No trouble has been experienced with adhesions.

Biesalski and Mayer,[6] who have made an extensive study of the gliding mechanism of tendons, have reached the conclusion that in order that the tendon may function well, the relationship between the tendon and sheath must be preserved. These surgeons have developed the tendon sheath exchange method, which recognizes the preservation of the normal relationship between the tendon and its sheath. This method is based upon physiological principles and has been excellently worked out.

Asepsis.—In performing the operation, the most rigid asepsis is required. The means of obtaining strict asepsis is generally appreciated and need not be described. Perfect hemostasis is required, and the Esmarch bandage is used in this work.

The Pull of the Tendon.—Some tension should be placed upon the transplanted tendon before it is inserted, so that when it is sutured with the limb in overcorrection, there is a moderate pull upon the tendon. Undue tension, however, should be avoided, since it might lead to the giving way of the subperiosteal attachment. In transplanting, a straight line of pull should be established from the origin of the tendon to its insertion. Care should be taken to avoid sharp angles or kinks. In case the tendon is found too short to meet the point destined for its insertion, it may be elongated by fascia lata or silk. The use of silk for this purpose has been worked out in detail by Lange.[7]

The Insertion of the Tendon. In the early transplantations, the lateral edges of the paralyzed tendon and the healthy tendon were anastomosed. Since a firm attachment was not assured by this method, other means of anchorage were proposed. Vulpius[8] introduced a procedure that involved severing the healthy tendon and sewing it to the paralyzed one. Lange[9] instituted a method of direct periosteal implantation. Several modifications of Lange's technique have been advocated, the most reliable of which is the subperiosteal or bone method of insertion, which consists of imbedding the tendon in a groove of bone and closing the periosteum over it. Union of the tendon and the bone is more certain if a small piece of bone is taken with the tendon upon its removal from its original attachment. The bone method of insertion is to be preferred in transplantations in the lower extremity. In the upper extremities where the strain is less, tendon-to-tendon attachments may be used satisfactorily. Any suture material used in transplantation should be buried under plenty of subcutaneous tissue.

After-treatment. The success of transplantations depends to a great extent upon the proper execution of the postoperative treatment. The more carefully and thoroughly the after-care is carried out, the greater is the possibility of securing a good result. The duration of the period of immobilization varies according to the part involved. When the plaster bandage is removed, some form of apparatus is usually worn to prevent strain and stretching until weight-bearing may be allowed with safety. Upon the removal of the plaster bandage, massage, exercises, and reëducation of the muscles are carried out under the supervision of a trained physiotherapist. The important feature of the after-treatment is teaching the patient to use the transplanted muscle. The postoperative treatment should be continued until function is reëstablished.

Tendon Transplantations in the Foot. There are only three or four conditions in the foot in which tendon transplantations may be considered, due to the fact that the anterior and posterior tibial muscles are usually affected, and there are no muscles that can be substituted satisfactorily for these strong groups.

Paralysis of the Peronei. *Talipes Varus.* The paralyzed peroneal group may be replaced satisfactorily by the tibialis anticus. The transplantation of this muscle into the cuboid is a measure far superior to the transplantation of the outer half of the Achilles tendon into the peronei, which is recommended by some operators. Such a procedure not only involves the splitting of the tendon, but also gives no stability, rarely increases function, and very materially weakens the gastrocnemius. In the opinion of the writer, this transplantation is to be discouraged.

Paralysis of the Tibialis Anticus and Posticus.—*Talipes Valgus.*—In paralysis of the tibial muscles with valgus deformity, transplantation of the peronei into the scaphoid has definite value when used as a supplementary measure to an astragalo-scaphoid arthrodesis. It is only in very rare cases that the transplantation alone is satisfactory, because the power of the transplanted peronei cannot replace that of the strong tibiales.

Extensive transplantations, consisting of the substitution of the extensor of the great toe for the tibialis anticus, and of the flexor of the great toe for the tibialis posticus, are to be discouraged. Such muscles cannot take the place of the powerful tibial groups.

Paralysis of the Tibialis Anticus.—*Toe-drop.*—In paralysis of the anterior tibial muscle with toe-drop deformity, transplantation of the extensor proprius hallucis has been recommended. This tendon lacks the qualifications necessary for a successful transplantation. The extensor hallucis, which is a long, slender tendon with very little power, cannot possibly be an adequate substitute for the anterior tibial muscle. Moreover, the removal of the extensor hallucis as a dorsal flexor of the great toe joint considerably lessens the stability of this joint.

A fixation operation is indicated in paralytic toe-drop.

Paralysis of the Gastrocnemius.—*Talipes Calcaneus.*—In calcaneus deformity resulting from involvement of the gastrocnemius, transplantation of the peroneus longus or of the tibialis posticus to either side of the Achilles tendon has been recommended by some operators. The writer believes that these transplantations give permanent results only when they are performed in conjunction with stabilizing procedures. In the use of operative measures in talipes calcaneus, secondary deformities must be taken into consideration; the foot is arched, the toes are contracted, and the astragalus is pushed forward—changes that result in shortening of the posterior fulcral arm of the foot. In transplantations, the full power of the muscles is not retained unless the posterior fulcral arm is lengthened. Astragalectomy or subastragalar arthrodesis produces this lengthening, and transplantation of the peronei when combined with these stabilizing operations increases the function of the foot.

Transplantation of the Tibialis Anticus into the Cuboid for Varus Deformity.—This is the most successful tendon transplantation performed in paralytic conditions of the foot. The results are consistently good. Before the transplantation is performed, deformity should be corrected by manipulation or by open operation. In some cases the transplantation may be done at the same time that a bone wedge is removed from the astragalus to correct the deformity, but in the experience of the writer the best results have been obtained when the deformity was corrected first, and the tendon transplantation performed several months later.

Operative Technique.—An incision (No. 1), about 2 inches long, is made over the attachment of the anterior tibial tendon, beginning about 2 inches above its insertion (Fig. 66). The tendon sheath is split and the attachment of the tendon exposed (Fig. 67). The tendon is then severed at its insertion, and if possible, the operator takes with the tendon a bit of its bony attachment (Fig. 68).

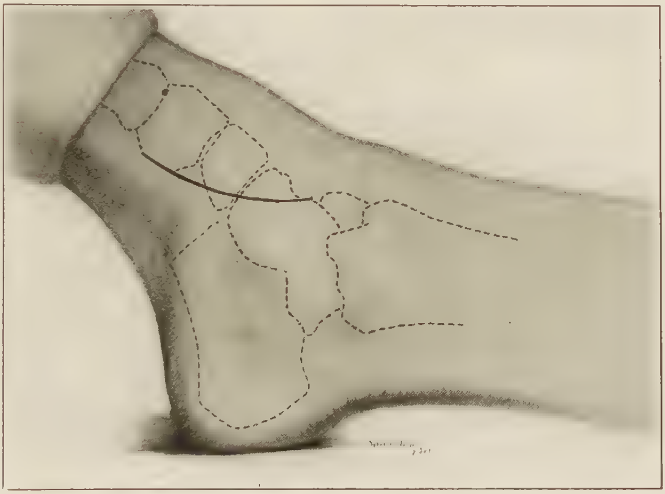

FIG. 66.—*Anterior tibial transplantation for talipes varus.* Incision (No. 1) over the attachment of the anterior tibial tendon.

A 2-inch incision (No. 2) is made over the anterior tibial muscle, beginning about 4 inches above the ankle joint (Fig. 69).

A semicircular incision (No. 3) is made over the cuboid (Fig. 69). The skin and fascia are elevated. A similar incision is made into the tissue of the cuboid, with the flap pointing in the direction of the anterior tibial tendon.

By means of a blunt dissector, the whole tendon is pulled through its sheath and drawn out of the second wound. The end of the tendon is quilted with chromic gut or linen. A pair of forceps is introduced into the cuboid wound and passed under the skin and fascia to the incision over the tibia. The sutures and the end of the anterior tibial tendon are grasped and pulled into the cuboid wound (Fig. 70). If desired, the anterior tibial tendon may be transferred through the sheath of the extensor longus digitorum (Biesalski and Mayer[6]).

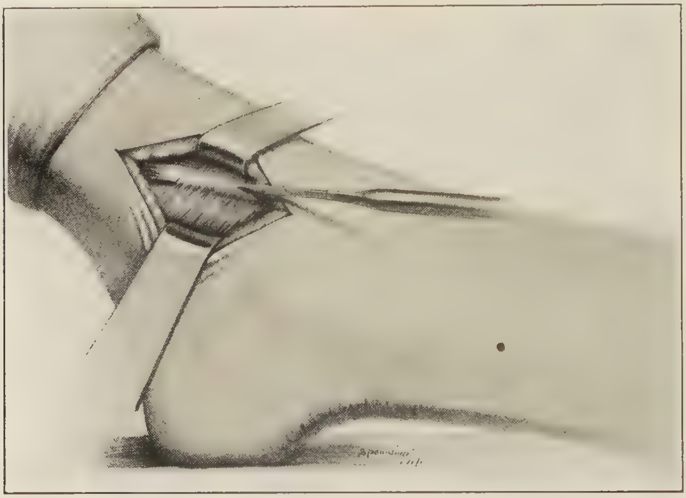

Fig. 67.—*Anterior tibial transplantation for talipes varus.* Exposure of the attachment of the tendon.

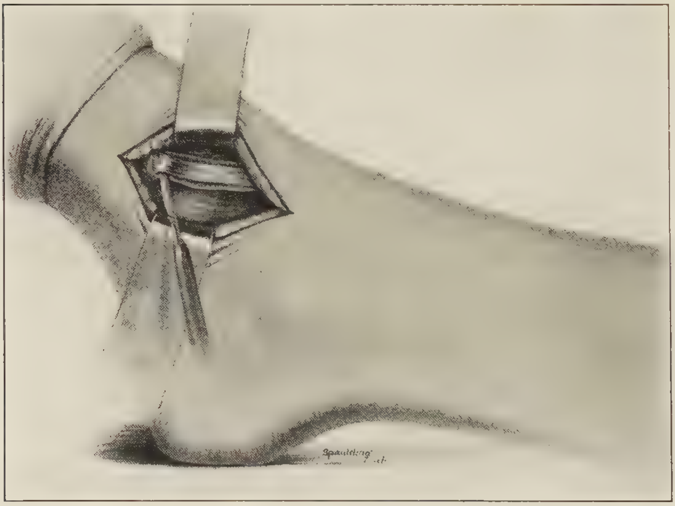

Fig. 68.—*Anterior tibial transplantation for talipes varus.* Severance of the tendon at its insertion.

The sheath of the anterior tibial tendon is closed with continuous fine catgut. The skin of wounds No. 1 and No. 2 is closed with continuous catgut.

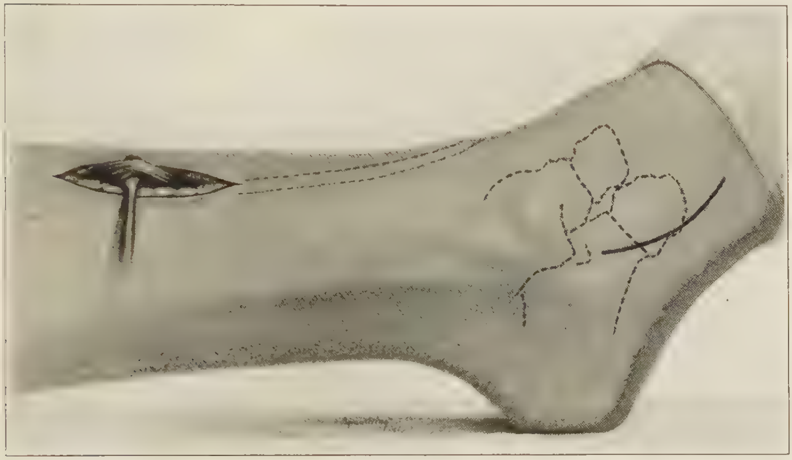

Fig. 69.—*Anterior tibial transplantation for talipes varus.* Incision (No. 2) over the anterior tibial muscle, and incision (No. 3) over the cuboid.

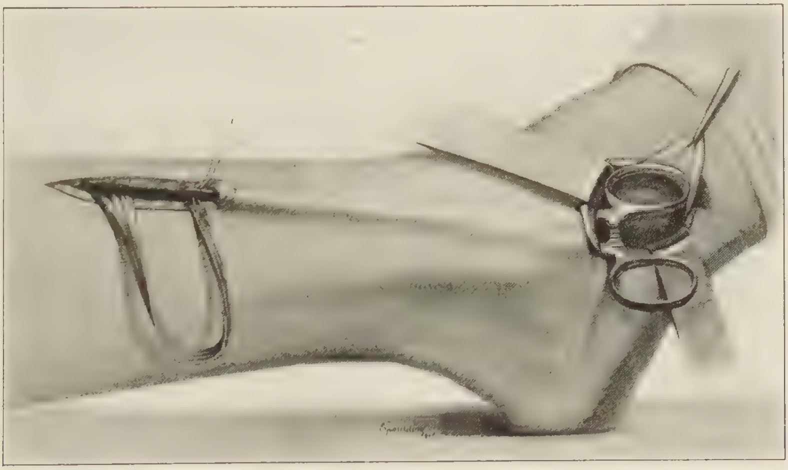

Fig. 70.—*Anterior tibial transplantation for talipes varus.* The anterior tibial tendon has been drawn through its sheath, and quilted with chromic gut. A pair of forceps is inserted to pull the tendon into the cuboid wound.

In wound No. 3, one of the tendon sutures is passed by means of the infantile hook into a cavity made in the cuboid, and brought out through the periosteum. The remaining suture is passed through the body of the cuboid, coming out on the opposite side. The foot is placed in an overcorrected position. The sutures are pulled tightly, bringing the tendon into the cuboid cavity (Fig. 71). The skin is closed with continuous catgut. A plaster bandage is applied from the toe to just above the knee, with the foot held at a right angle and in the position of valgus.

After six weeks, the plaster is split to allow the foot to receive local treatment. Passive and active movements are begun early, and as soon as sensitiveness has subsided, weight-bearing on the cast is encouraged. Plaster bandages are worn for eight to ten weeks, changes being made, if necessary, after the third or fourth week. Upon the removal of the last plaster bandage, a varus brace is applied and worn for about six months. Later, a raise on the outer side of the sole of the shoe assists in maintaining correction. It is well to maintain the foot in overcorrection and prevent any strain on the transplant for one year.

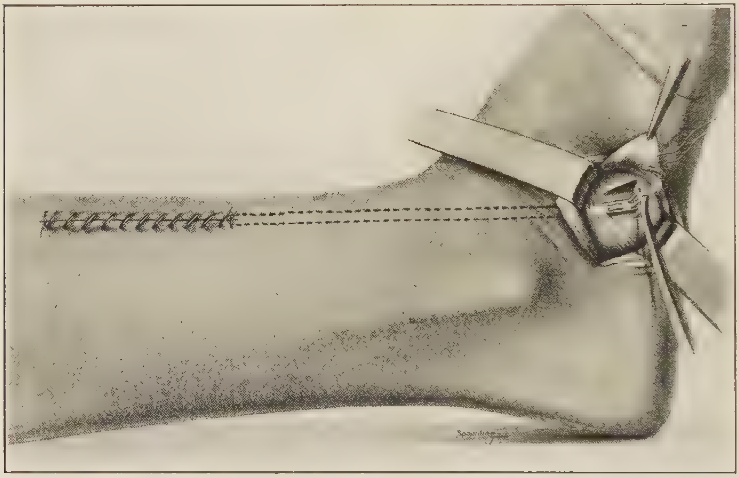

FIG. 71.—*Anterior tibial transplantation for talipes varus.* Insertion of the anterior tibial tendon into the cuboid.

Transplantation of the Peronei into the Scaphoid for Valgus Deformity.—This operation is used most successfully as a supplementary measure to an arthrodesis of the astragalo-scaphoid joint. One or both peroneal tendons may be transplanted as required. The results are satisfactory, except that atrophy of the calf muscles persists.

An incision (No. 1) is made over the tendon of the peroneus longus, beginning about 5 inches above the tip of the fibula and extending to the attachment of the peroneus brevis into the cuboid (Fig. 72). The sheath is split, and the tendon severed as near its attachment as

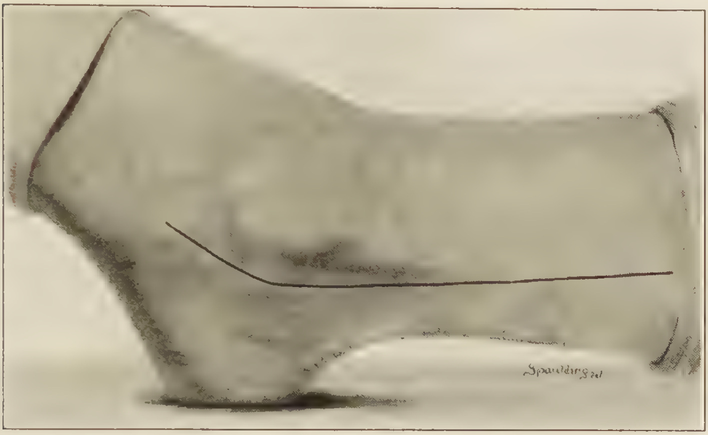

Fig. 72.—*Peroneal transplantation for talipes valgus.* Incision (No. 1) over the body of the peroneus longus.

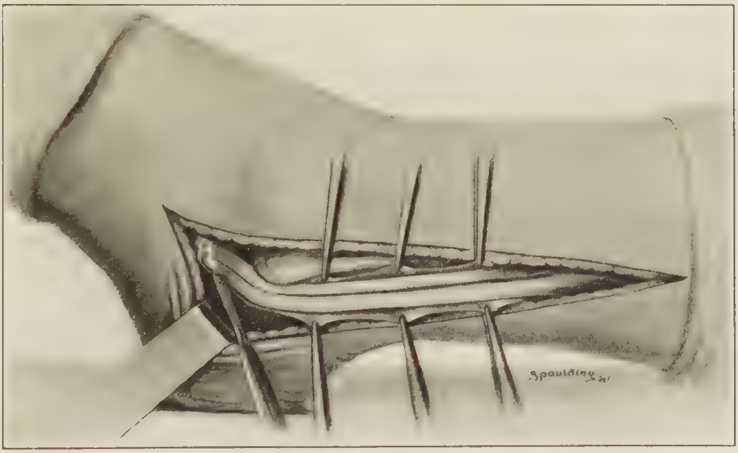

Fig. 73.—*Peroneal transplantation for talipes valgus.* Exposure of the peroneal tendons.

possible (Figs. 73 and 74). If both peronei are to be transplanted, the same procedure is carried out, and the tendons are sutured together for several inches with interrupted silk.

A second incision (No. 2) is made over the attachment of the anterior tibial tendon into the scaphoid. This incision should be curved, with the flap facing downward toward the heel. A wedge of bone is elevated from the scaphoid, with the flap pointing toward the ankle-joint. The groove thus prepared will receive the peroneal tendon (Fig. 75).

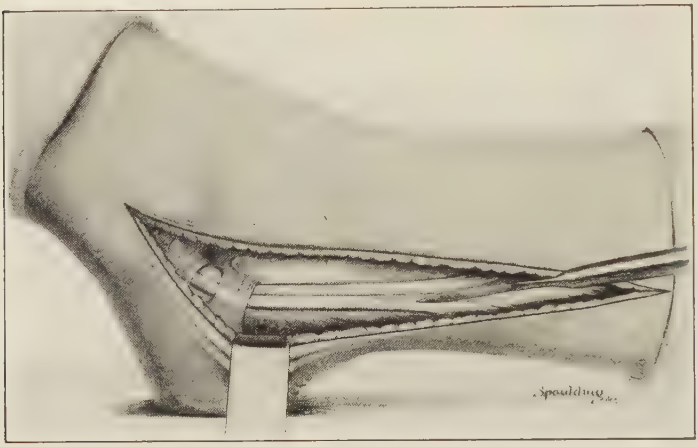

FIG. 74.—*Peroneal transplantation for talipes valgus.* Splitting of the tendon sheath.

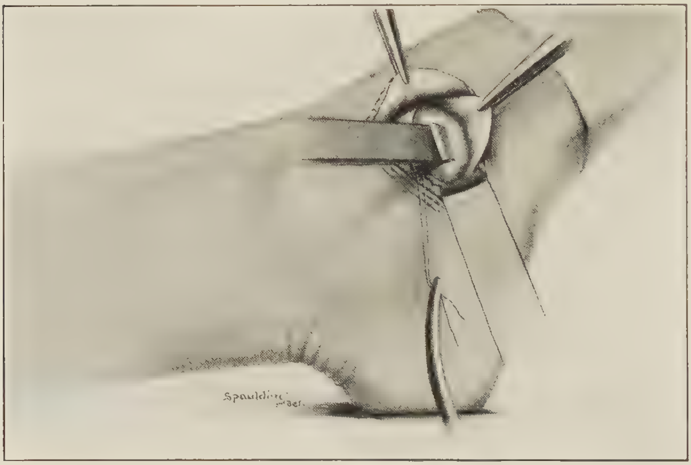

FIG. 75.—*Peroneal transplantation for talipes valgus.* Making a groove in the scaphoid to receive the peroneal tendon.

The peroneal tendon is quilted with chromic gut or linen. A pair of long dressing forceps is inserted under the skin and fat from wound No. 2 to wound No. 1, and spread to allow sufficient space for the passage of the tendon. The tendon is then passed under the fat and fascia and pulled into the cavity made in the scaphoid (Fig. 76). In so far as possible, the tendon should pull in a direct

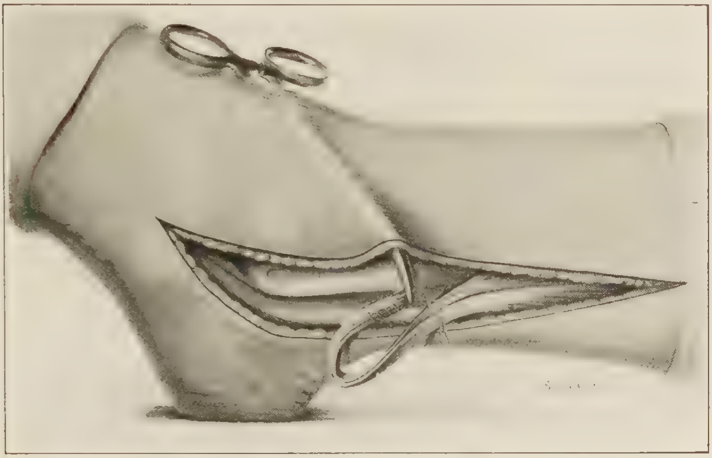

FIG. 76.—*Peroneal transplantation for talipes valgus.* The sheaths of the peroneal tendons have been split, the tendons severed near their attachment, and quilted with chromic gut. The forceps are being inserted to pull the tendon into the scaphoid wound.

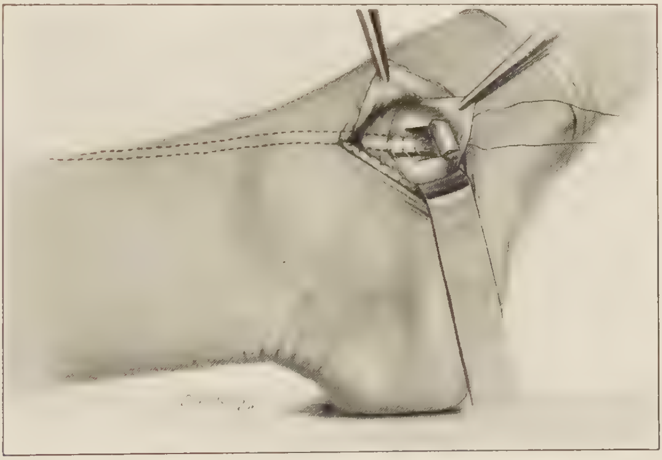

FIG. 77.—*Peroneal transplantation for talipes valgus.* Insertion of the peroneal tendon into the groove in the scaphoid.

line. It is sometimes possible to slip the peroneal tendon through the sheath of the paralyzed anterior tibial, but in the majority of cases it is simpler to pass the tendon under the fat and fascia. Wound No. 1 is closed with continuous catgut.

During the insertion and suture of the tendons, the foot should be held in varus and slight dorsiflexion. The infantile hook is threaded with one of the tendon sutures, and this is passed through the scaphoid and brought out through the flap at its base. The second suture is passed in a similar direction, a short distance from the first. The tendon is then pulled into position and the periosteum is sutured over the tendon ends. The two tendon sutures are then tied tightly (Fig. 77). Wound No. 2 is closed with continuous catgut. A plaster bandage is applied from the toes to just below the knee, with the foot in the position of varus and at a right angle.

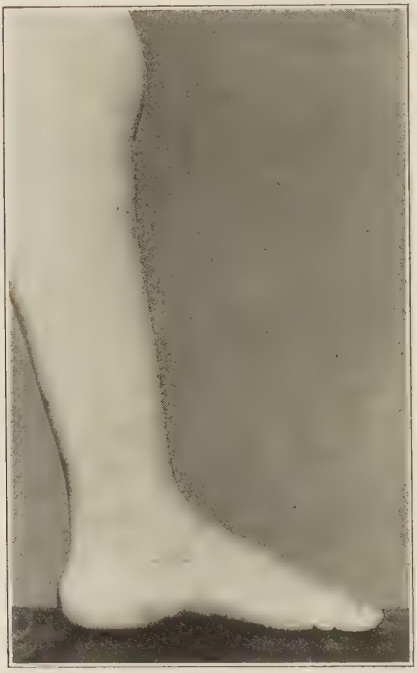

Fig. 78.—Case VIII. D. M. *Talipes valgus.* Lateral view ten years, nine months after arthrodesis of the astragalo-scaphoid joint and transplantation of the peroneus longus into the scaphoid.

The first plaster bandage is worn for six weeks, and then changed. The patient usually begins walking when the new plaster is applied. Two weeks after application it is split, and massage and exercise are carried out daily. The foot is maintained in overcorrection during the first three months after operation by means of plaster bandages, and during the following year, a Whitman brace with a lift on the inner side of the sole and heel of the shoe is worn.

Case VIII.—D. M., aged four years.

Complaint: Paralysis of the left leg.

Duration: Two and a half years.

Cause: Infantile paralysis.

Physical Examination: The left leg was mottled and cold. The calf and thigh were markedly atrophied and there was ½ inch shortening. The sartorius and quadriceps had good power. The anterior tibial muscle had no power. The patient walked with eversion of the leg and with the foot in valgus.

Treatment: *May 7, 1912.* An infantile valgus shoe was fitted. Although this brace held the foot in fair correction, the patient walked with the foot in slight valgus.

December 3, 1912. An arthrodesis of the astragalo-scaphoid joint and a transplantation of the peroneus longus into the scaphoid were performed.

Result: *September 23, 1923.* The patient had a very stable foot. The valgus was fully corrected (Fig. 78).

Transplantation of the Peroneal Tendons into the Achilles Tendon.—This operation may be performed in young children who show a tendency to develop calcaneus deformity. It serves to strengthen the posterior muscle group, to retard rapid development of cavus, and to prevent the forward displacement of the foot, so common in calcaneus deformity. The power of plantar flexion is increased by this operation.

Transplantation of the peroneal tendons is commonly used as a preliminary measure to a stabilizing operation. In older children, it may be done in conjunction with an astragalectomy to increase balance. Both the peroneus longus and brevis are used in transplantation.

Operative Technique.—An incision is made over the peroneus longus, beginning at the cuboid and extending to about 2 inches above the external malleolus. The sheaths of the peroneus longus and brevis are opened, and the tendons divided below the external malleolus and retracted.

A longitudinal slit is made in the Achilles tendon through which the peroneal tendons are passed and sutured. The tendons are then united with their distal stumps. During this fixation, the foot should be held in plantar flexion (Fig. 106). The skin is closed with continuous catgut No. 2. A plaster bandage is applied from the toes to the knee, with the foot in plantar flexion.

The plaster is worn for eight to ten weeks. Weight-bearing is permitted about the second or third week. In six or eight weeks the plaster bandage is split, and massage and light exercise are begun. Some form of retentive apparatus is worn for about a year to prevent the strain of active use.

Transplantation of the Common Extensor and the Extensor Hallucis for Claw-foot (Forbes[10]).—This operation is preceded by a subcutaneous plantar fasciotomy with forcible manipulation of the forefoot. If the cavus is excessive, it may be necessary to correct it by a wedge-shaped osteotomy through the dorsal surface of the metatarsals or astragalus.

Operative Technique.—A horse-shoe incision is made on the dorsal surface of the foot. The incision is carried down to the extensor tendons, and a flap containing all tissues superficial to them is turned back on the dorsal surface of the tarso-metatarsal articulation. The long extensor tendons are detached from the phalanges and transplanted into the necks of the metatarsal bones.

Anchorage is secured by boring transversely through the neck of the metatarsals or obliquely from each side, or by passing a silk ligature around the metatarsal head, carrying the ligature from the dorsal to the plantar surface by means of a needle which is made to hug the neck of the metatarsal bone. Strong pressure must be exerted beneath the metatarsals to raise them. The ligature is then returned from the plantar to the dorsal surface of the foot by inserting the threaded needle into the same perforation of the plantar skin as that through which it made its exit. The needle is made to hug the opposite side of the metatarsal bone, and drawn out at almost the same spot as that on the dorsal surface into which it was originally thrust.

The silk attachments are buried deeply by fine catgut sutures, and the skin flap returned and sewed in place by horsehair.

Case IX.—W. W. (Courtesy of Dr. Mackenzie Forbes.)

Age: Thirteen years.

History: In 1916, the patient had an attack of poliomyelitis, resulting in a deformity of the right foot. He walked on the toes of this foot.

Examination: April 19, 1926. The right foot was held in the position of equinus and cavus with hyperextension of all the toes (Fig. 79). The patient could flex his foot, but only to an angle of

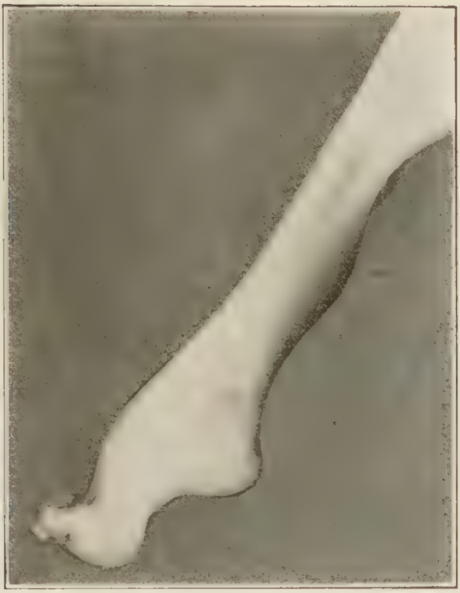

Fig. 79.—Case IX. W. W. *Equinocavus deformity.* Before treatment, showing the foot in equinocavus deformity, with hyperextension of the toes. (Courtesy of Dr. Forbes.)

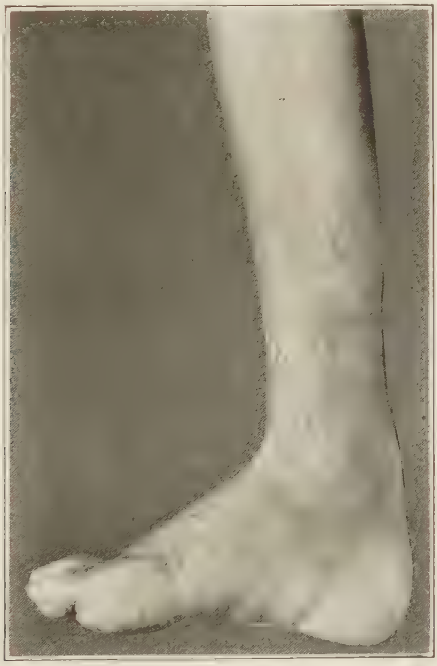

Fig. 80.—Case IX, W. W. *Equinocavus deformity.* After treatment, showing foot in good position. (Courtesy of Dr. Forbes.)

about 45 degrees. There was hyperextension of all the toes when the foot was flexed. Extension of the foot was possible. The tendo Achillis and plantar fascia were shortened. There was subluxation of the heads of the metatarsal bones on the dorsal aspect.

Treatment: The Achilles tendon was lengthened; the plantar fascia was lengthened at the os calcis; the extensor tendons were transplanted; a wedge was removed from the dorsal surface of the first metatarsal; and an interphalangeal arthrodesis was performed (Fig. 80).

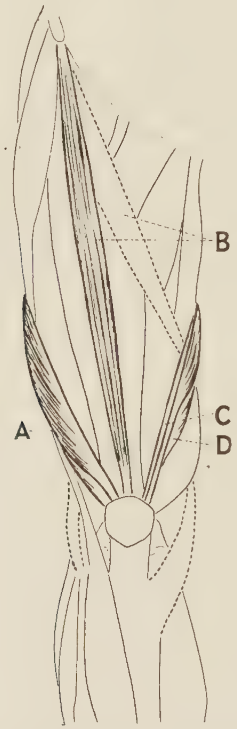

Fig. 81.—Anterior view. Transplantation of the sartorius, biceps, semimembranosus and semitendinosus into the patella. *A*, biceps; *B*, sartorius; *C*, semimembranosus; *D*, semitendinosus.

Tendon Transplantation in the Knee (Fig. 81).—When the quadriceps is paralyzed, transplantations may be performed to increase the stability of the knee. By the transference of one of the flexors forward, the extensor power is slightly increased and the flexor power diminished, thus making the knee more stable. In many cases the patient no longer feels the necessity of placing his hand on his thigh while walking. In general, these transplantations have little functional value, although some voluntary extension may be obtained if any power remains in the quadriceps.

There are several muscles available for transplantation, and operators are not agreed as to the value of each. The writer believes that the sartorius lends itself most satisfactorily to transplantation. Fortunately, this muscle usually escapes paralysis. One or both hamstrings may also be used for transplantation. The tensor vaginæ femoris is another muscle available, but as it adds external support to the knee, it is better not to make use of it unless imperative. Care must be taken to leave one good flexor in position, and to make sure that the gastrocnemius, which acts as a flexor, is functioning.

The after-treatment in knee transplantations is important. Particular care should be taken to avoid strain by protecting the limb for at least six months; the plaster bandage is worn for six weeks following the operation, and bandages or other forms of protection are worn during the remaining period. Three weeks after the operation, weight-bearing on the plaster is permitted. Six weeks after the operation, local therapy, including, massage, gentle manipulations, and exercises, is begun.

Transplantation of the Sartorius into the Patella.—A curved incision is made on the inner thigh, beginning at a point about half-way between the groin and the patella, passing over the top of the patella, and ending on the outer side of the knee. The skin and fat are strongly retracted. The sartorius is cut at a point opposite the patella and freed from its surrounding muscles and tissue as far as the mid-thigh. The end of the tendon is quilted with chromic or linen sutures. A slit is made in the lower quadriceps tendon just above the patella through which the sartorius is drawn and brought to the patella, where it is attached subperiosteally. The sartorius is sutured to the quadriceps by interrupted catgut sutures for a distance of 3 inches. The wound is closed with continuous catgut sutures, and a plaster bandage applied from the ankle to the groin, with the knee in extension.

Case X.—D. M., aged seventeen years.
Complaint: Unable to walk without crutches.
Duration: Two years.
Cause: Infantile paralysis.
Physical Examination: In the right leg, the peronei were absent, the anterior tibial muscle and the gastrocnemius were present, and

the quadriceps had slight power. In the left leg there were no peronei; the anterior tibial muscle and the quadriceps had slight power (Fig. 82).

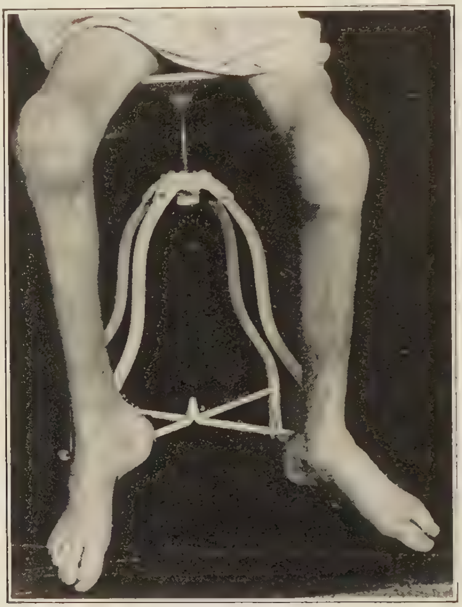

FIG. 82.—Case X. D. M. *Paralysis of the quadriceps.* Before treatment—no power of extension in either leg.

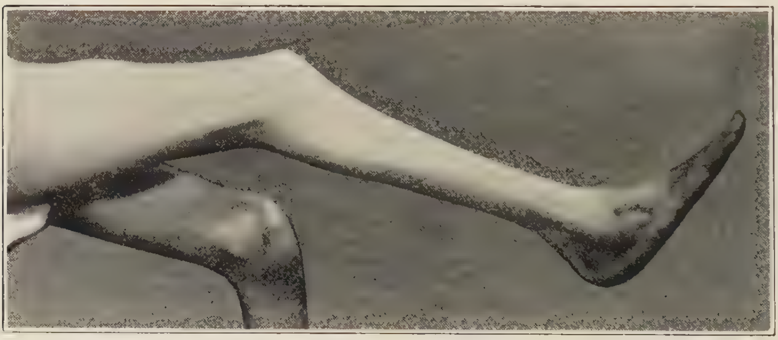

FIG. 83.—Case X. D. M. *Paralysis of the quadriceps.* Showing the power of extension in the right leg, thirteen years after transplantation of the sartorius.

Treatment: April 26, 1913. A transplantation of the sartorius was done in the right leg, and the foot was manipulated into valgus.

September 11, 1913. An astragalectomy of the left foot was performed.

May 22, 1914. A V-shaped wedge was removed from the right foot to correct the varus deformity.

February 27, 1915. In the left leg the tensor vaginæ femoris was transplanted into the patella.

October 1, 1917. A supracondylar osteotomy of the left femur was performed to correct the knock-knee deformity.

Result: The result from the sartorius transplantation was excellent. (Fig. 83) shows the power of extension of the leg, thirteen years after transplantation of the sartorius.

Transplantation of the Biceps into the Patella.—An incision is made on the outer side of the leg, running parallel to the tendon of the biceps, and extending from the mid-thigh to a point near the upper quadriceps pouch; the incision then curves over the top of the patella, and finally terminates on the inner side of the knee. The skin and fat are separated and retracted. The biceps tendon is severed as near as possible to its attachment to the fibula. The tendon is freed from the surrounding muscles and tissue attachments as high as possible, usually as far as the mid-thigh. The end of the tendon is quilted with chromic gut. It is then drawn through a slit in the quadriceps tendon and inserted subperiosteally into the patella.

The biceps is sewed to the quadriceps tendon with several interrupted sutures. The wound is closed with continuous catgut sutures. A plaster bandage is applied from the ankle to the thigh, with the knee in extension.

When paralysis of the sartorius is present, transplantation of the biceps may be combined with transplantation of the semitendinosus and semimembranosus.

Transplantation of the Semimembranosus and the Semitendinosus into the Patella.—An incision is made on the inner side of the leg, beginning midway between the knee and the groin, and running parallel to the thigh until it reaches a point opposite the top of the quadriceps pouch; the incision then curves anteriorly to the outer side of the patella. The skin and fat are separated and retracted. The semimembranosus and semitendinosus are severed near their insertion, and separated from their surrounding attachments as high as possible. The ends of the tendons are quilted with chromic or linen sutures. A slit is made in the lower quadriceps just above the patella, through which the tendons are drawn. They are then carried to the top of the patella and fastened subperiosteally.

The wound is closed with continuous catgut sutures. A plaster bandage is applied from the ankle to the thigh, with the knee in extension. The plaster is worn for two or three months.

Transplantation of the Tensor Vaginæ Femoris into the Patella.—An incision is made on the outer side of the thigh, beginning about midway between the patella and the anterior-superior spine, curving forward, and ending at a point between the inner side of the patella and the internal condyle. The skin and fascia are separated and retracted. Two incisions, about 2½ inches apart, are then made in the fascia lata, starting parallel and gradually converging until they meet at the outer condyle. The strip of fascia thus formed, attached at its base, is turned toward the patella. A transverse incision is then made in the periosteum over the patella, and the fascia lata fixed subperiosteally. The fascia lata is sewed along the quadriceps tendon with interrupted sutures for a distance of 3 inches from the patella. The skin wound is closed with continuous catgut. A plaster bandage is applied from the ankle to the groin, with the knee in extension.

Tendon Transplantations in the Hip.—Tendon transplantations in the region of the hip are limited practically to cases of weakened gluteus medius and minimus. In properly selected cases, these operations are effective and result in considerable improvement of the gait. Their efficacy depends upon the following factors: The gluteus maximus must be active; some power must be present in the gluteus medius; and the lateral abdominal muscles must be strong.

It is questionable whether the favorable results obtained from transplantations about the hip are due to the transference of power, or to the return of activity in the weakened muscle after a period of rest. Cases have come to the writer's attention, in which the recovery of power in the paralyzed muscle, and not the transplant, was responsible for the increase in function.

Transplantation of the Tensor Vaginæ Femoris (Method of Legg[11]). —The incision starts over the anterior-superior spine, extends backward and downward over the great trochanter, and then passes downward along the course of the femur for about 3 inches. The fascia is incised downward from the anterior-superior spine to 3 inches below the great trochanter, where it is divided transversely backward for 1½ inches. The fibers of the vastus externus are divided to expose the femur. A periosteal flap is turned downward, and a groove about 1 inch long and ½ inch wide is made into the marrow. The free end of the fascia is sutured with No. 18 twisted silk and inserted into the groove by carrying the silk ends through holes drilled in the femur on either side of the groove. The sutures are knotted over the fascia in the groove, and the periosteal flap is turned back and sutured over the groove.

Before the suturing is completed, the thigh is abducted about 30 degrees, and the fascia is placed under moderate tension.

The skin and subcutaneous fat flap is turned back and sutured. A plaster spica is applied from the waist to the ankle, with the leg in 30 degrees abduction. Massage is begun after two weeks, and muscle training after four weeks. The patient begins to walk on crutches about the fourth week. The spica is worn for two months, and after its removal an abduction splint is worn for six months.

Legg has performed this transplantation in 25 cases, and satisfactory results were obtained in the majority.

Transplantation of the Vastus Externus (Method of Lange[12]).—A longitudinal incision, beginning in the middle of the thigh and carried up to the trochanter, frees the upper half of the vastus externus. Another incision, down to the bone, is made on the crest of the ilium from the anterior-superior spine to the anterior edge of the gluteus maximus. The upper end of the vastus externus is freed from the great trochanter. The muscle is then interlaced for 6 to 8 cm. with 8 or 10 strands of heavy silk. The silk cords are passed subcutaneously to the upper wound. One strand of silk is fixed behind the spine, the second is fastened a few centimeters farther back, and so on, so that the silk tendons radiate from the upper end of the vastus externus to the crest of the ilium.

Periosteal insertion is done when possible. In places where the periosteum is very thin, holes are bored in the rim of the iliac crest, and the silk drawn through them. While the suturing is being done, the leg is held in 140 degrees abduction.

The skin wounds are closed, drainage is obtained by a sublimate wick (for forty-eight hours), and the leg is fixed in plaster in extreme abduction. The cast is removed after six or eight weeks, and a supportive apparatus worn to fix the leg in about 150 degrees abduction. Active abduction movements are begun against resistance. About four months after operation, the patient may begin walking without support.

The Substitution of the Erector Spinæ for Paralyzed Gluteal Muscles.—Lange suggested the use of a part of the erector spinæ muscle as a substitute for the paralyzed glutei. He has used this procedure in approximately 50 cases and has obtained satisfactory results (Steindler[1]).

Kreuscher[13] has worked out a method based upon Lange's suggestion. He has reported one case in which this method was used successfully; complete stability was restored to the hip-joint, and definite gluteal action was obtained.

Operative Technique (Kreuscher).—"The patient is placed face downward upon the operating table. The lumbar spine is elevated by placing sand-bags under the abdomen to make the erector spinæ muscle more accessible. An incision about 8 inches long is made over the lower portion of the left erector spinæ muscle, beginning at the posterior crest of the ilium, upward along the long axis of the muscle. The muscle sheath is exposed and split longitudinally and the outer one-half of the erector spinæ is detached at the crest of the ilium and freed upward for about 6 inches. The sheath of the muscle is closed, leaving the detached portion lying upon it. Two series of silk sutures each containing 4 strands are placed in the prepared stump of muscle from above downward in such a way as not to interfere more than necessary with the blood and nerve supply. The sutures are left sufficiently long to reach well beyond the greater trochanter, their point of future fixation. A short curved incision with the convexity forward is next made, exposing the greater trochanter and a portion of the upper end of the shaft of the femur. A 12-inch forceps is then carried from this incision tunneling its way through the fat adjacent to the gluteal muscular sheath upward and medially until its tip passes out of the upper incision and grasps the silk sutures which have been placed in the erector spinæ muscle. Through this fatty tunnel the sutures are drawn down and are ready for fixation. Two of them are passed through a drill hole which has been made through the greater trochanter and two are carried around the shaft of the femur just above the lesser trochanter. At this stage of the operation the leg is brought in extreme abduction and the silk is tied with sufficient tension to draw down the stump of the erector spinæ muscle but not so taut as to necrotize the muscle fibers. To provide a smooth sliding surface for the silk a piece of fascia lata $1\frac{1}{2}$ by $2\frac{1}{2}$ inches is sutured over the posterior aspect of the crest of the ilium. Thus we avoid a destruction of the silk strands as they pass over the iliac ridge. This accomplished, another tunnel is made from the greater trochanter directly upward to the crest of the ilium. To these two fixed points two additional silk sutures are attached to aid in the abduction of the leg. In all incisions the skin is closed without drainage. The entire procedure must be carried on with the strictest aseptic technique as even the slightest infection would necessitate the immediate removal of the silk and, in consequence, a failure of the operation. After the completion of the operation a leg and body cast is applied, and care is used to keep the leg in extreme abduction.

"The silk which is used is sterilized by boiling to insure its asepsis. It is furthermore rendered antiseptic by placing it for twenty-four hours in a 20 per cent aqueous solution of oxycyanide of mercury.

"The abduction cast is permitted to remain on for eight weeks, during all of which time the patient is encouraged to exert abduction action. At the end of two months the cast is removed and the legs are placed in a Travois or similar abduction splint. This position is assumed for at least six months, or until such time as the silk has become securely attached and sufficiently organized to withstand the subsequent strain. The patient is then permitted to be up and about on crutches, being warned never to adduct the leg beyond the straight line of the body. Massage and electrotherapeutic measures are carried on over the entire extremity and mild electrical stimulation over the erector spinæ muscle for the purpose of strengthening it and developing it to the fullest degree."

Tendon Transplantation in the Wrist and Fingers. – **Transplantation of the Pronator Radii Teres, Flexor Carpi Radialis, and Flexor Carpi Ulnaris in Extensor Paralysis** (Jones[14]). –*Operative Technique.* –Two large incisions are made: One, J-shaped, is made over the back and outer side of the lower part of the arm, the J extending up the radial side as high as the insertion of the pronator radii teres, and the hook crossing the back of the carpus and ending on the middle of the posterior aspect of the wrist-joint; the other incision, a vertical one, is made over the anterior aspect of the lower part of the flexor carpi ulnaris. The pronator radii teres is freed from the radius. The flexor carpi radialis and the flexor carpi ulnaris are cut close to their insertions. The wrist and fingers are now placed in complete dorsiflexion and the thumb in abduction, and these positions are maintained during the remainder of the operation. The radiocarpal extensors are slit longitudinally, and the pronator radii teres inserted and sutured. The suturing is performed in only one-half of each tendon to prevent loss of blood supply in the tendon below the suture.

The flexor carpi radialis is inserted into the three extensors of the thumb and into the extensor of the index finger, and the flexor carpi ulnaris into the extensors of the remaining three fingers to produce extension. During the entire procedure of tendon suture, the wrist and fingers are kept in complete dorsiflexion and the thumb in full abduction, in order to procure the best action from the transplanted tendons. After operation, the extremity is placed in a splint which keeps the hand in full dorsiflexion, with the metacarpo-phalangeal and interphalangeal joints flexed at an angle of approximately 10 degrees.

Transplantation of the Pronator Teres, Palmaris Longus, Flexor Carpi Radialis, and Flexor Carpi Ulnaris. (Modification of Stiles[15]).—Stiles obtains dorsiflexion of the wrist by transplanting the pronator teres into the extensors carpi radialis longior and brevior. Extension of all three joints of the thumb is secured by transplantation of the palmaris longus into the secundi internodii pollicis, and of the flexor carpi radialis into the extensor primi internodii pollicis and the abductor pollicis longus. When the palmaris longus is absent, one-half the tendon of the flexor carpi radialis is substituted for it. The extension of the fingers at the metacarpo-phalangeal joints is obtained by transplantation of the tendinous portion of the flexor carpi ulnaris into the tendons of the extensor communis digitorum, the extensor indicis, and the extensor minimi digiti. The ulnaris tendon is threaded through a slit made in each tendon and fixed by a linen thread suture. Lastly, the fleshy half of the distal part of the flexor carpi ulnaris is sutured to the extensor carpi ulnaris to prevent radial deviation of the hand during dorsiflexion.

Transplantation of the Flexor Carpi Radialis, Flexor Digitorum Sublimis, and Flexor Carpi Ulnaris (Method of Stoffel[16]).—Stoffel recommends transference of the flexor carpi radialis to the extensor carpi radialis brevior, transference of the flexor digitorum sublimis to the abductor pollicis longus and the extensor pollicis brevis, and transference of the flexor carpi ulnaris to the extensor digitorum communis and the extensor pollicis longus.

Method of the Writer for Transplantation of the Radial and Ulnar Tendons in Extensor Paralysis.—The attachment of the flexor carpi radialis is severed through a 1-inch incision made directly over it on the anterior surface of the wrist. A second incision is made half-way up the forearm along the course of the flexor carpi radialis tendon. The tendon is then pulled up (Fig. 84). A long L-shaped incision is made across the back of the wrist and up the side of the ulna. The flap is dissected upward. The tendon of the flexor carpi radialis is thrust obliquely over the edge of the radius and made to appear over the back of the lower end of the radius. The ulnar tendon is severed at its attachment, carefully dissected upward as far as the incision permits, and the whole muscle turned over, so that the tendon lies along the back of the lower end of the ulna.

During the completion of the operation, the wrist and fingers are held in hyperextension. The radial and ulnar tendons are crossed through slits made in the extensors (Fig. 85). If no provision has been made for extension of the thumb by the use of the pronator radii teres or the palmaris longus, the radial and ulnar tendons are woven through the extensors of the thumb. The end of the radial tendon is then sutured to the ulnar tendon on the ulnar side, and the end of the ulnar tendon is attached to the radial tendon on the radial side.

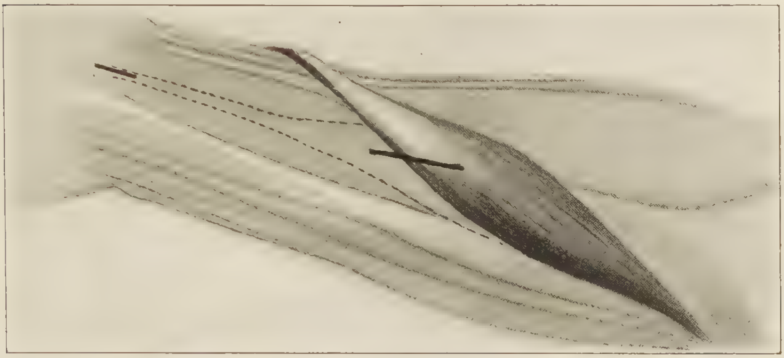

FIG. 84.—*Tendon transplantation in the hand.* Showing incisions over the attachment of the flexor carpi radialis and along the course of the tendon. The tendon is thrust obliquely over the edge of the radius.

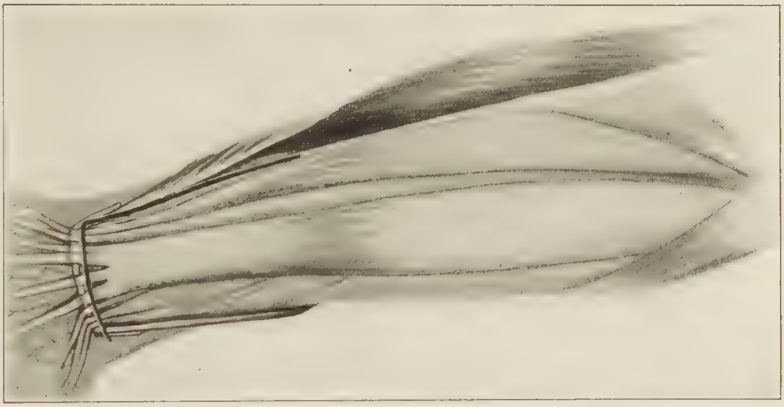

FIG. 85.—*Tendon transplantation in the hand.* Showing the L-shaped incision across the back of the wrist and up the side of the ulna. The ulnar tendon has been severed at its attachment and the whole muscle turned over. The radial and ulnar tendons are seen running through the extensors which are slit to receive them.

The wound is closed. A splint is applied with the wrist held in hyperextension. This is worn for six to eight weeks. Massage and exercises are carried out in the after-treatment.

Case XI.—F. A.

Complaint: No power to extend wrist (Fig. 86).

Treatment: November 5, 1919. A tendon transplantation was performed according to the above technique.

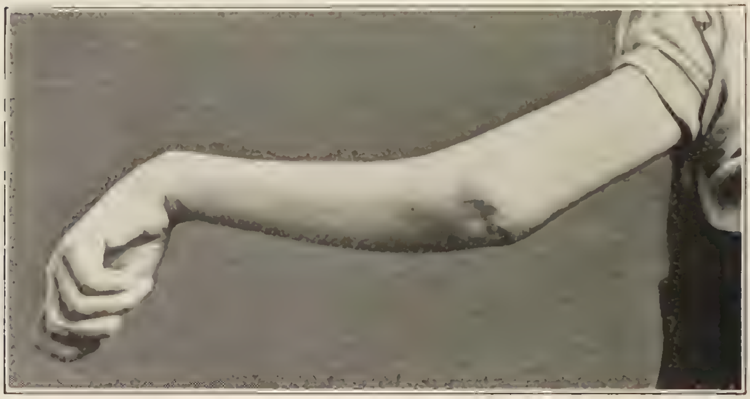

Fig. 86.—Case XI. F. A. *Paralysis of the extensors of the hand.* Showing wrist-drop and hand deformity before tendon transplantation.

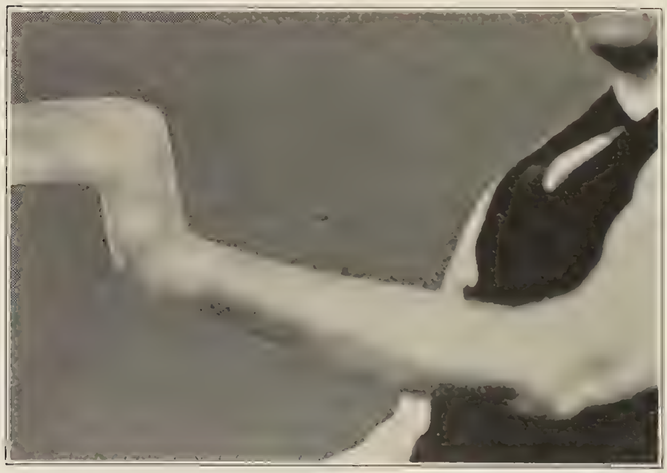

Fig. 87.—Case XI. F. A. *Paralysis of the extensors of the hand.* Voluntary extension four years after tendon transplantation.

November 29, 1919. Massage and muscle training were started.

Result: September 8, 1920. The patient had voluntary extension of the wrist to within 15 degrees of normal; voluntary extension of the thumb was deficient, but satisfactory. Fig. 87 shows the power of voluntary extension, four years after operation.

Transplantation of the Extensor Radialis Longior and Suturing of the Flexors in Median Nerve Paralysis (Jones[17]). -*Operative Technique.* - "A vertical incision is made, about 3½ inches long, from the upper border of the anterior annular ligament parallel to the length of the arm and close to the inner side of the radial artery. Another incision parallel to this one and of 4 inches in length is made at the same level on the back of the radius over the extensor carpi radialis longior tendon. The extensor carpi radialis longior is now divided close to its insertion, and the tendon is freed from fascia to the upper limit of the incision. It is now passed under the bridge of skin and brought to the anterior aspect of the arm. The thumb being flexed slightly into the palm, the transplanted tendon is sutured to the flexor longus pollicis. The line of suture here should not be thick because any marked increase in bulk in the tendon at the line of suture prevents its working in the sheath which lies on the anterior aspect of the thumb. Through the same anterior incision the flexor sublimis tendons and the median nerve are now retracted inward, and after making the adjacent sides of the flexor profundis rough, lateral suture of the outer two to the inner two tendons is performed."

Transplantation to Replace the Long Flexors of the Thumb, Index Finger, and Middle Finger (Stiles[15]).—Preceding the transplantation, the thumb is arthrodesed at its carpo-metacarpal joint in apposition and abduction, so that the angle between the first and second metacarpals is 60 degrees.

The extensor ossis metacarpi pollicis is detached as near its insertion as possible, and one-half of the tendon transplanted into the tendon of the flexor longus pollicis at the wrist. Flexor power is restored to the terminal phalanx of the thumb by this transplantation. The power of the flexors of the index finger is replaced by transplanting the extensor carpi radialis longior. This tendon is detached from its insertion and brought to the front of the forearm in a subcutaneous tunnel superficial to the supinator longus and then deep to the tendon of the flexor carpi radialis. The tendon is split longitudinally, and one-half of it transplanted into the sublimis and one-half into the profundus tendon of the index finger just above the anterior annular ligament. This transplantation should be done with more than semi-flexion of the interphalangeal joints.

Substitution of the Opponens Pollicis by the Long Flexor of the Thumb (Steindler[1]).—*Operative Technique.*—"The incision is made on the volar side of the thumb from the middle of the end-phalanx down to the thenar running along the ulnar border of the long flexor of the thumb. The incision should not reach beyond the middle of the thenar in order to save such thenar branches of the median nerve as have escaped paralysis. The branches for the opponens pollicis, the abductor brevis and the radial half of the short flexor of the thumb are given off at the point lying in the proximal half of the thenar eminence. The long flexor of the thumb is now exposed, its sheath is incised and the edges of the sheath are held by fine forceps. The tendon is lifted out of its sheath and split longitudinally in two halves. The outer or radial half of the tendon is severed at the distal end of the split, reflected upward and then the sheath of the long flexor is reunited over the remaining ulnar half of the tendon. The radial tendon flap is carried around through a tunnel made in the soft parts. It is led to a point at the base of the basal phalanx well on its dorsal surface. A short incision is made at this point through which the tendon is led out and here it is fastened to the periosteum of the basal phalanx of the thumb. The wounds are closed. The hand is now dressed with the thumb in opposition and is left in this position for four weeks when drill exercises of the thumb are started.

"This procedure simply provides a second attachment for the flexor of the thumb, so that when this muscle contracts, the tendon flap brought around the thumb, causes it to swing around in opposition with the trapezius as a pivot. The thumb is then able to approach the little finger and to carry out gripping motions."

Transference of the Short Extensor of the Thumb (Method of Ney[18]). —*Operative Technique.*—"A dorsal incision, extending from the metacarpo-phalangeal joint to 3 cm. above the attachment of the extensor ossis metacarpi pollicis, exposes the short extensor of the thumb. The sheath of the short extensor tendon is opened, and the tendon is dissected to a point about 3 cm. above the insertion of the extensor ossis metacarpi pollicis. It is here divided. The tendon is now wrapped with wet gauze and the palmar surface of the hand is exposed.

"The palmar incision is made over the prominence of the palmaris longus tendon and palm for about 9 cm. The sheath of the palmaris longus tendon is opened and the tendon divided at a point where it begins to spread out into the palmar fascia. The tendon is wrapped in gauze and held back over the wrist. The dorsal and palmar incisions are connected subcutaneously by a channel. The extensor brevis pollicis tendon is grasped by the forceps and pulled through the channel to the palmar incision. It is pulled under the anterior annular ligament to the bed of the palmaris longus tendon. The pulling on this transposed tendon now occupying the new position will produce a typical opposing action of the thumb. With the thumb held in this position and the hand flexed on the wrist the transposed tendon is united to the tendon of the palmaris longus.

"The former sheath of the palmaris longus is now closed over the transplanted tendon and the palmar and dorsal skin incisions closed. The hand and wrist should be retained in flexion—the thumb occupying its new position—by suitable bandaging and splinting. The hand and forearm should remain splinted in this manner for about one month. After two months, splinting may be discarded.

"Before operation, the patient should be taught how to contract his palmaris longus muscle. Two weeks after operation, he should again start contracting the palmaris longus."

After this operation, the long flexor of the thumb can work advantageously, and the patient is able to pick up objects.

TENODESIS.

The conversion of paralyzed tendons into ligaments, for the purpose of restoring stability and balance to an unstable and distorted foot, has a place in the operative field of paralysis. In varus, valgus, and equinus deformities, in which stabilizing operations or tendon transplantations are not indicated, tenodesis may be used to advantage. The fixation operation has more definite value if performed in conjunction with a stabilizing operation, since a paralyzed tendon seldom can withstand the strain of the body-weight and the pull of the powerful opposing muscle groups.

Tenodesis is contraindicated in talipes calcaneus and in flail-foot. Stabilizing procedures are always required to restore balance in such cases. If a tendon fixation is performed preliminary to a stabilization, posterior displacement of the foot, which is the essential feature of a stabilizing operation, is accomplished with difficulty.

The use of tendons as ligaments has several advocates. Reiner,[19] Codivilla,[5] Vulpius,[8] Jones,[20] Gallie,[21] and others have described this method of stabilization. Several methods of securing anchorage of the tendon have been suggested: Codivilla introduced the subperiosteal attachment; Vulpius sutured the tendon to a flap of fascia and to the periosteum; Gallie attempted to secure more thorough fixation by making the tendon fast in a groove in the bone. Gallie's technique is in general use today. His method was devised originally to overcome talipes varus, but later was developed for application to other paralytic deformities.

Operative Technique in Talipes Varus (Gallie, Fig. 88).—A vertical incision $2\frac{1}{2}$ inches long is made over the external malleolus, exposing the bone and peroneal tendons.

The sheaths of the peroneal tendons are split, and the tendons carefully freed. They are then split into strands by longitudinal incisions, or scarified by numerous small transverse cuts. This scarification is done to ensure a firm union between the tendon and underlying bone.

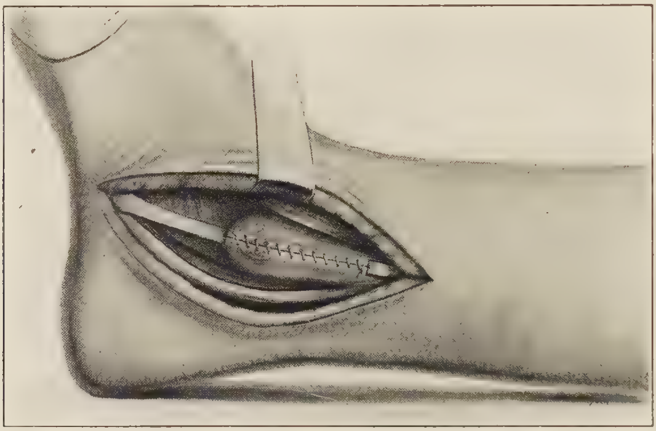

FIG. 88.—*Gallie fixation operation for talipes varus.* Fixation of the peronei into the fibula.

A longitudinal incision is made through the periosteum on the outer aspect of the external malleolus and shaft of the fibula, extending down past the epiphyseal cartilage to the tip of the bone. The periosteum is raised for $\frac{3}{16}$ inch on either side of this incision, and if the epiphysis consists of cartilage, a flake of it is raised with a knife to correspond to the reflected periosteum. A trough is then gouged out of the bone for the entire length of the incision, and of sufficient depth to allow one of the tendons to be buried in it. A similar trough is prepared on the posterior surface of the malleolus adjacent to the groove normally occupied by the tendon. The deformity of the foot is then entirely overcome by manipulation, and the foot is held in a slightly overcorrected position by an assistant. With the aid of a pair of Kocher clamps, the tendon of the peroneus longus is drawn taut and laid in the anterior groove.

At the lower end of the incision, a medium-sized kangaroo tendon suture is passed twice through the tendon and cartilage of the epiphysis on either side of the groove, and firmly tied over the tendon.

The assistant now releases his hold on the foot to test the solidity of the fixation, as it is very important to have the proper amount of tension on the tendon. The periosteum is sewed over the tendon with No. 1 catgut.

The peroneus brevis is similarly buried in the posterior groove. The wound is closed with catgut and horsehair, and a plaster-of-Paris bandage applied from the toes to the knee. The cast remains on for six weeks to two months, weight-bearing being allowed.

The important detail of the operation is the burying of one tendon in front of the malleolus and the other behind it, to make the tendon balance secure and thus avoid any tendency to equinus or calcaneus. The peroneus brevis, which is the main abductor of the foot, functions more effectively when buried behind the malleolus. When all suturing has been completed, the foot should be held in the exact position desired; slackness or over-tautness of the tendons should be avoided.

The same principles of operation are applied to equinus and valgus deformities. The best results in equinus deformities are obtained when some power remains in the muscles of the toes. When both the dorsiflexors and the peronei are involved, the anterior tibial tendon is sewed to the anterior surface of the tibia at the front of the ankle, and the proximal end of the tendon of the peroneus longus is threaded through a hole drilled in an antero-posterior direction through the lower end of the fibula. Care should be taken to free the tendons of the tibialis anticus and peroneus longus from the annular ligaments, so that the tendons run in a straight line from their insertion to the point of fixation. This step will avoid any sagging of the foot later from stretching of the annular ligament.

In talipes valgus, tenodesis is of most value when combined with an astragalo-scaphoid arthrodesis. In exceptional cases the tenodesis alone may be used. When the valgus deformity is associated with equinus, the tibiales posticus and anticus are utilized in operation. The tendon of the tibialis anticus is drawn through a hole drilled in an antero-posterior direction through the internal malleolus, and fastened to the tibialis posticus, which has been placed in a groove cut subadjacent to the normal position of the tendon. Another method of fixation is to prepare two grooves in the malleolus, widely separated at the lower extremity of the bone and meeting about

1½ inches higher on its inner surface, and to suture the tendons, which have been clamped together, over the point of bone formed at the union of the two grooves.

Case XII (author's).—E. B., aged twenty years.
Complaint: Pain in right ankle.
Duration: Six or seven months.
Cause: Old infantile paralysis.

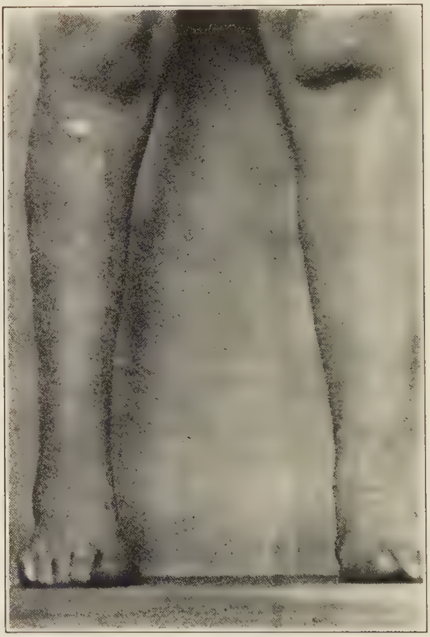

Fig. 89.—Case XII. E. B. *Talipes equinovarus.* Five years after fixation of the peroneal and dorsiflexor muscles.

Physical Examination: The posterior tibial muscle was strong. The peroneal and anterior tibial muscles were paralyzed. The foot was in the position of equinovarus. The gastrocnemius was powerful.

Treatment: January 19, 1921. A Gallie fixation of the peronei and outer dorsal flexor muscles as well as a tenotomy of the Achilles tendon was performed.

Result: Eight months after operation, the patient walked well with a brace. There was no toe-drop.

March 15, 1922. One year and two months after operation, the patient was walking very well without braces. The foot was straight. Since that time the foot has continued to grow stronger, and the fixation has held. The patient walks with an almost imperceptible limp.

Fig. 89 shows the result *March, 1926,* five years after operation.

The Whitman Loop Operation for Equinovalgus.—This operation was devised by Whitman[22] to be used in the correction of equinovalgus deformity, when all the muscles about the ankle, except one or both tibiales, are active. The presence of power in the other muscles is very necessary to the attainment of a successful result; otherwise, implantation of the anterior tibial tendon may produce varus or calcaneus deformity.

The purpose of the operation is to overcome the abducting force by removing the peronei brevis and tertius, to increase the power of dorsiflextion by transplanting the peroneus brevis and the hallucis longus to the inner border of the foot, and to decrease the tendency to deformity by displacing the dorsal tendons from the outer to the inner side of the foot. Fixation of the tendons is accomplished by hooking about them the tendon of the tibialis anticus and implanting it in the tibia to act as further support for the inner border of the foot.

Operative Technique.—The technique of the operation is described by its originator as follows: "All restriction to adduction and dorsal flexion is overcome as a preliminary measure. This usually requires division of the tendo Achillis. An incision is then made behind the external malleolus, extending upward over the fibula to the junction of the lower and middle thirds. The common sheath of the peronei is opened, and the tendon of the brevis is freed from its attachment about the malleolus. Its insertion at the base of the fifth metatarsal bone having been cut through a small incision, the tendon is drawn back and its muscular fibers are separated from the fibula up to the point of transference. A long incision is then made over the center of the ankle extending from about 3 inches above the joint, downward to the metatarsal region. The tendon of the peroneus tertius, which restricts inward displacement, is cut at its insertion and separated from the adjoining tendons of the common extensor. These tendons are carefully dissected from the underlying tissue and the annular ligament is cut through so that they may be displaced to the inner border of the foot without tension. The tendon of the tibialis anticus is divided at its junction with the muscular substance and is drawn downward from out the sheath through a small opening near its insertion. The tendon of the peroneus brevis is passed through the sheath of the tibialis anticus according to the Biesalski-Mayer technique, and sewed to the attachment of the tibialis anticus and to the periosteum. The longus hallucis tendon is cut at the same plane, drawn through an opening in its sheath, and it, together with the tendon of the peroneus tertius, is attached to the periosteum of the scaphoid. The tendon of the tibialis anticus is then passed from within outward under all the dorsal tendons and is drawn upward and embedded in a groove, running in a direction upward and inward, cut in the inner border of the tibia after the Gallie method at a sufficient tension to hold the foot at a right angle to the leg and slightly inverted. The incisions are closed with two layers of catgut, the first for the repair of the tendon sheaths, and the second for a subcutaneous covering of the tendons. A plaster splint is then applied fixing the foot in the position above described."

Weight-bearing is allowed as soon as tenderness permits. The splint is usually worn three months, although it may be replaced before this time by a removable support that will permit exercise of the transplanted muscles. In late convalescence the inner side of the shoe may be raised, or a Whitman brace applied. Exercise, and stretching into dorsal flexion are very important in the after-treatment to obtain a full range of motion.

BONE OPERATIONS.

Stabilizing Operations of the Foot.—We have already called attention to the inadequacy of tendon transplantation, either as a means of maintaining correction of deformity or as a means of replacing the lost function of a paralyzed muscle. Although such procedures may result in temporary relief, the transplanted tendons often stretch, and deformity is likely to recur. To ensure permanent correction and stability of the foot, the joint balance itself must be restored, so that the foot can support the body-weight without strain.

The type of stabilizing operation applicable to a given case depends upon the age of the child and upon the extent of the muscle involvement. When it is possible to use more than one method in a case, the choice of operation rests with the surgeon.

In general, stabilizing operations should not be performed before the child is ten to twelve years of age. It also is not advisable to operate until two years have elapsed from the onset of the disease. During the interval, care should be taken to hold the deformity in check, either by means of supportive apparatus or by minor operative measures.

Stabilizing operations are not indicated in paralytic involvement of the foot, when other weight-bearing joints are seriously affected. The indication for each stabilizing measure will be discussed in detail under the operative procedure.

Astragalectomy.—The Whitman[22] operation of astragalectomy is accepted in America as the standard method of stabilization of the foot in children between the ages of eight and fifteen years. This operation not only ensures stability of the foot, but it has the great advantage of preserving motion.

The Object.—The object of astragalectomy is to make a new ankle-joint (an arthroplasty) which, with such muscle as is present, will be stable and well balanced. When properly done, an astragalectomy, by displacing the tibia and fibula forward, shifts the weight-bearing line to the front of the foot, thereby obtaining a so-called "rocking-horse foot" with the weight-bearing line near the middle the point of advantage—rather than on the "backstep of the rocking horse" as is clinically seen in a case of calcaneovalgus. The fulcrum is increased by lengthening the posterior arm, that is, by increasing the distance between the weight-bearing line of the tibia and fibula and the back of the heel. This change in weight-bearing is particularly efficient when the peronei are transplanted into the Achilles tendon, in which position they tend not only to prevent recurrence of calcaneus, but also to assist plantar flexion.

Indications and Contraindications.—Before astragalectomy is performed, deformity should be corrected by manipulative methods, and the foot should be held in overcorrection for several months. During this period weight-bearing is allowed.

Astragalectomy is indicated in calcaneus deformities, and especially in talipes calcaneovalgus, for which condition Whitman originally devised the operation. Astragalectomy may be indicated in calcaneovarus deformity, but the varus element must first be eliminated by repeated stretchings and retention of the foot in overcorrection for several months.

In flail-foot, astragalectomy establishes stability by transferring the weight to the center of the foot, by elongating the arch, and by making the os calcis more horizontal. If only the plantar flexors remain, great care must be taken to prevent the development of an equinus deformity.

In talipes equinus with no power remaining in the anterior group, astragalectomy is useful in increasing stability, but precaution must be taken to prevent the recurrence of deformity. In severe equinovarus, when the varus element has been corrected by repeated stretchings, the removal of the astragalus is frequently the *coup-de-grâce* needed to make the foot serviceable.

Astragalectomy is indicated in equinovalgus if other muscle groups in addition to the tibiales are weak. The "loop" operation, also devised by Whitman, is occasionally used in equinovalgus, when all the muscles about the ankle, with the exception of one or both tibiales, are active.

The operation may be indicated in varus or valgus deformity when the remaining muscle groups are weak. It also may be used in progressing varus or valgus deformity, which is not amenable to other procedures.

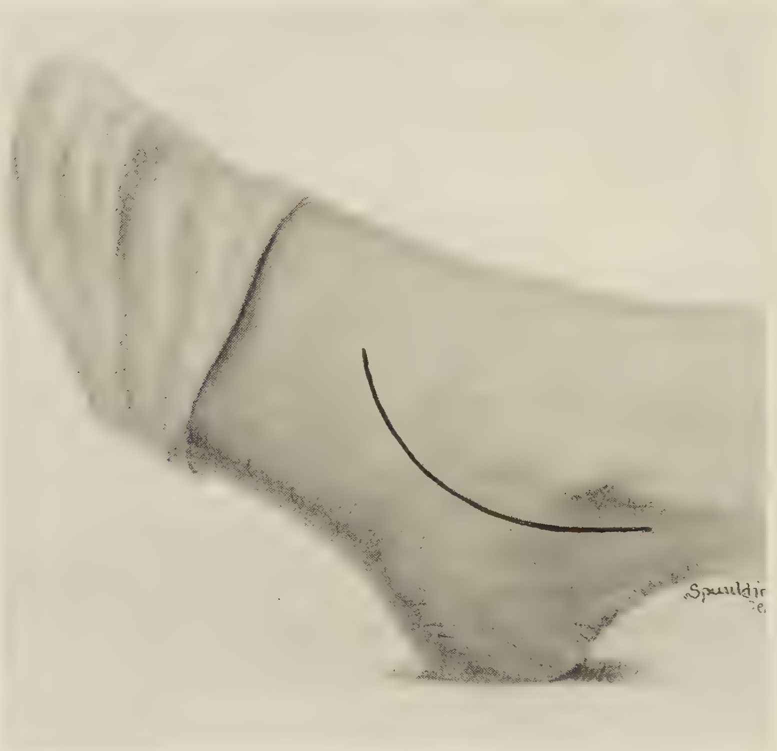

Fig. 90.—*Astragalectomy. The Whitman operation.* Line of incision.

Astragalectomy is very valuable in stabilizing a severe claw-foot. The extreme relaxation of the soft tissues, resulting from the removal of the astragalus, gives the opportunity to save many feet which might have required amputation.

Age.—The ages between eight and fifteen years have been found best for astragalectomy, since motion is preserved easily during this period. Stabilization often is not secured in cases operated before the age of eight, and in patients more than fifteen years of age, sensitiveness and pain may follow the astragalectomy.

Operative Technique.—Asepsis should be guarded carefully. All manipulations should be gentle. A tourniquet is applied after the Esmarch bandage has been adjusted.

A curved or L-shaped incision is made around the external malleolus, extending forward over the head of the astragalus (Fig. 90). The flap is dissected upward, exposing the tendons of the peroneus longus and brevis (Fig. 91). The tendons are severed at the fibular tip, their ends sutured with No. 2 catgut, and retracted (Fig. 92). An incision is then made through the external ligaments around the astragalus, special care being taken in cutting about the head of the astragalus (Fig. 93). The foot is inverted strongly. By placing a blunt dissector under the neck of the astragalus, the bone is pried out of position head first and removed, a veritable *coup-de-main* when well done, taking less than thirty seconds (Fig. 94). The ease

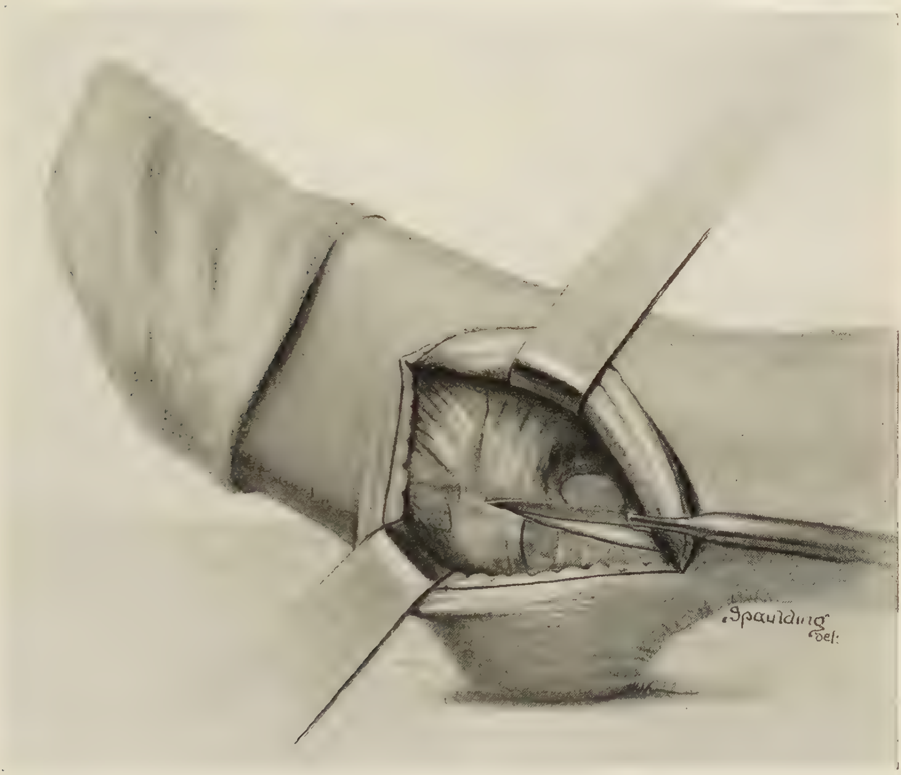

FIG. 91.—*Astragalectomy. The Whitman operation.* Tendon of the peroneus longus exposed; tendon of the peroneus brevis being exposed.

with which the foot may be displaced backward will be appreciated at once, but in order to ensure a stable joint, a certain amount of careful modeling must be done.

The internal lateral ligament is dissected upward from the internal malleolus. If the case is one of equinovarus, the dissection should be carried farther up than in other cases. If necessary, a strip of cartilage is removed from the tip of the tibio-fibular articulation to allow spanning of the scaphoid and cuboid (Figs. 95 and 96). A thin slice of bone is then excised from the sides of the scaphoid and cuboid to form pockets for the malleoli (Fig. 97). The ligaments are dissected to make a pocket for the fibula by the cuboid (Fig. 98). The foot is displaced backward and held carefully in this position to ensure the proper relations in the new joint. The backward displacement of the foot, which checks lateral movements by the formation of actual bony contact between the scaphoid and the tibia,

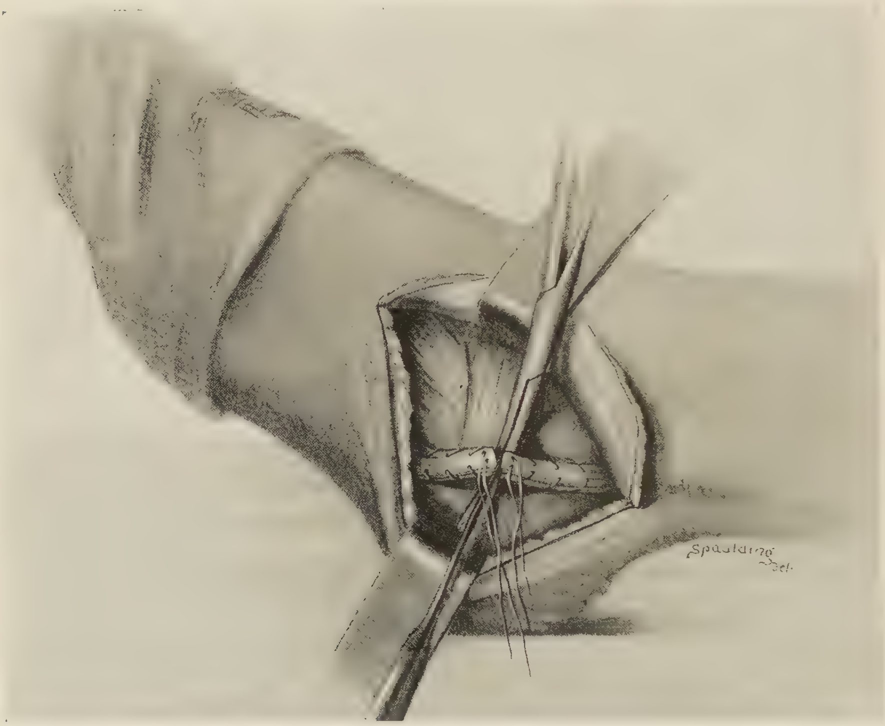

Fig. 92.—*Astragalectomy. The Whitman operation.* Division of the peroneal tendons and suturing of their ends.

is the essential feature of the Whitman operation. A moderate equinovalgus position ensures stability while holding (Figs. 99, 100, 101, and 102). The peronei are disposed of by (*a*) suture to the Achilles tendon, by (*b*) tendon fixation to the fibula, or by (*c*) resuture to the tendons of the peronei, as indicated in the given case (Figs. 103, 104, 105, and 106). The skin is closed with catgut, and a plaster bandage applied from the toes to the mid-thigh with the knee flexed and the foot in slight equinovalgus (Fig. 107).

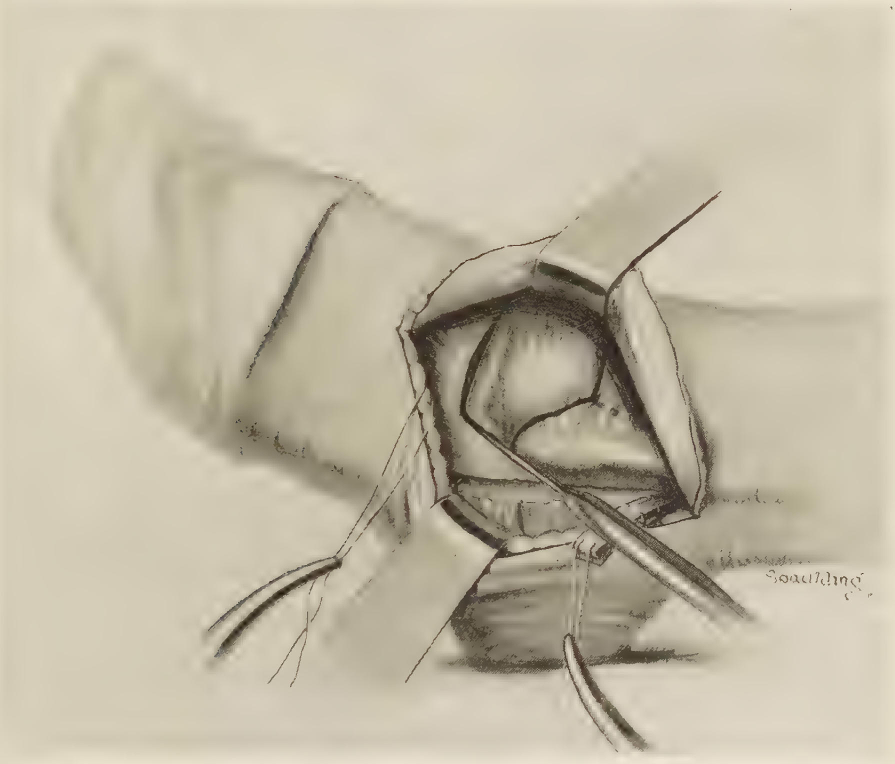

Fig. 93.—*Astragalectomy. The Whitman operation.* The external ligaments around the astragalus have been divided. The astragalus is being defined.

The after-care should be carefully supervised by the operator. The leg is kept elevated for a period of ten days. Walking with crutches may then begin. The first change of the plaster bandage takes place in three or four weeks, at which time the equinus is corrected to a right angle if the quadriceps is present, or is left in 5 to 8 degrees equinus in cases in which the quadriceps is paralyzed.

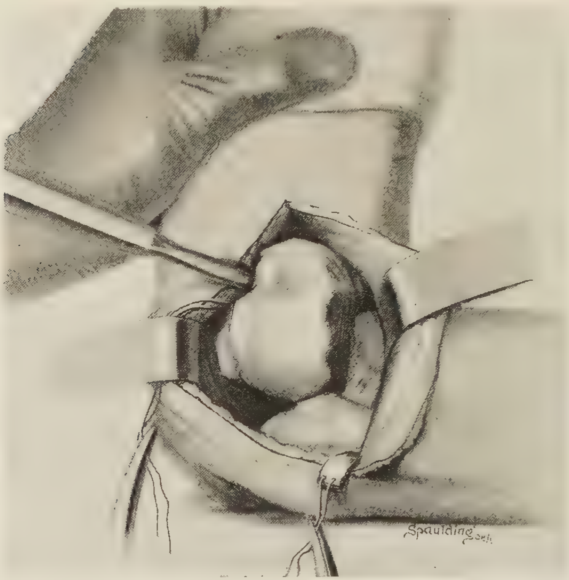

Fig. 94.—*Astragalectomy. The Whitman operation.* Dislocation of the astragalus by inversion of the foot. Note the heavy dissector under the neck, by which the astragalus is pried loose head first.

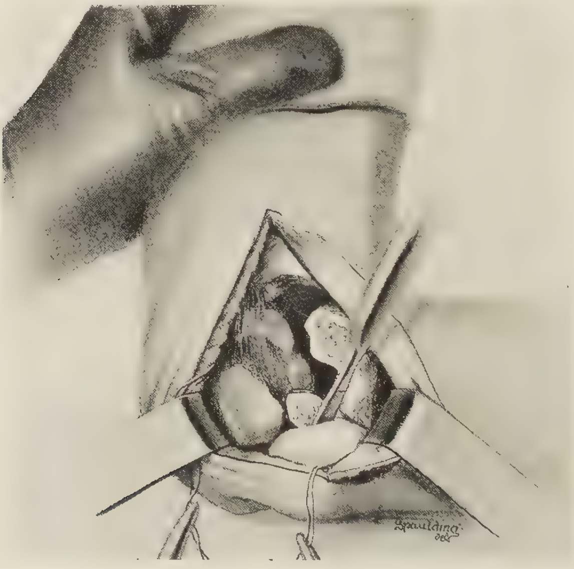

Fig 95.—*Astragalectomy. The Whitman operation.* Widening the tibio-fibular articulation by the removal of a strip of cartilage from the external malleolus.

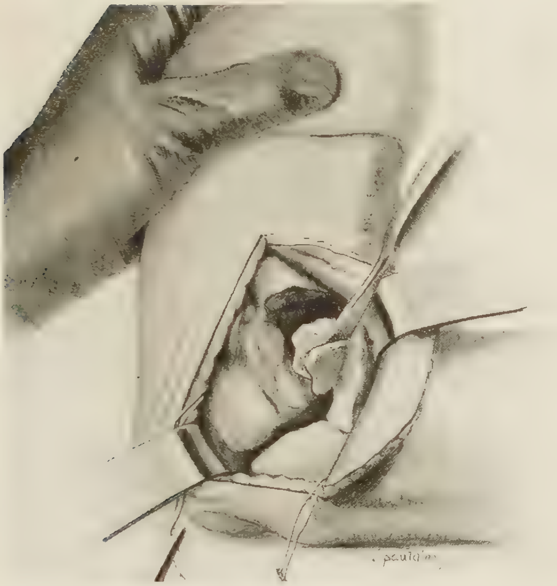

Fig. 96.—*Astragalectomy. The Whitman operation.* Widening the tibio-fibular articulation by the removal of a strip of cartilage from the internal malleolus.

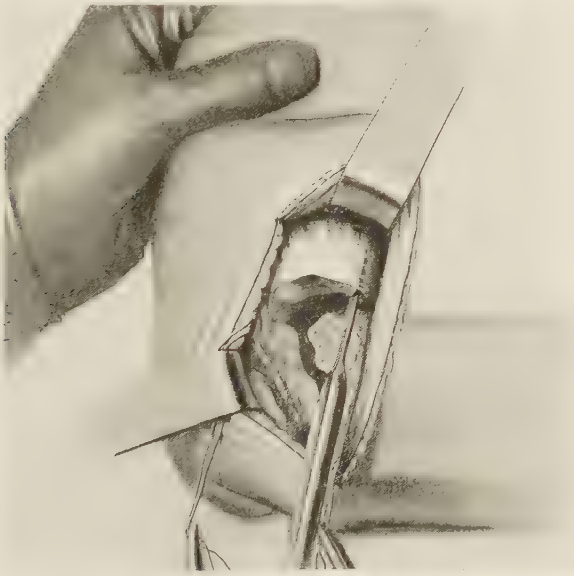

Fig. 97.—*Astragalectomy. The Whitman operation.* The new articulation for the internal malleolus. A thin slice of bone has been removed from the scaphoid to form a pocket for the sharpened internal malleolus.

The mechanical value of this step is apparent. Valgus should persist to a slight degree throughout the treatment.

About four weeks after operation, a leather shoe is put over the plaster, and the child is allowed to walk. The plaster is changed when necessary during the next five or six months. After the removal of the last plaster, a shoe with a lift on the outer side of the sole is worn to maintain the foot in valgus. A $\frac{1}{2}$ or $\frac{3}{4}$ inch cork lift worn under the heel will compensate for the shortening of the leg and improve the gait. It is sometimes advisable for older patients to wear a limited-motion ankle brace in the form of an orthopedic shoe with either an inner or outer upright, to protect the foot from strain during the early months of weight-bearing.

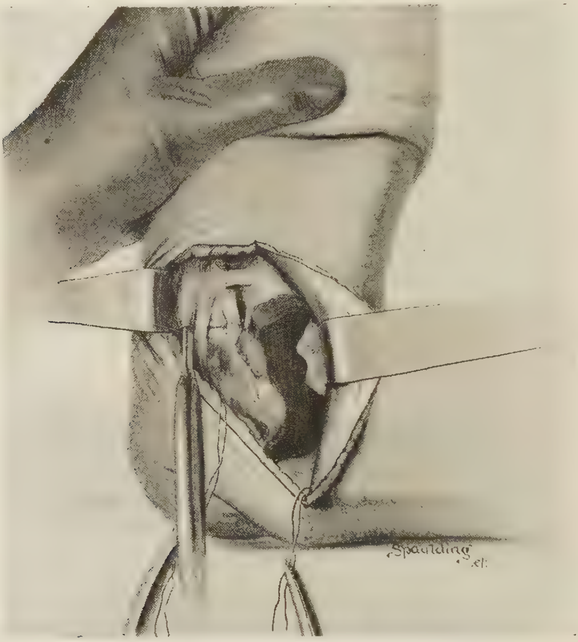

Fig. 98.—*Astragalectomy. The Whitman operation.* Dissection of the ligaments to make a pocket for the fibula by the cuboid.

With the return of active use, circulation improves rapidly, loss of growth ceases, and the paralyzed leg begins to grow at a rate equal to that of the well side.

Results.—The writer has used this operation in over 400 cases. It has been possible to follow up and examine 247 of these patients. The majority of the cases were calcaneovalgus deformities, but varus, equinovarus, equinovalgus, and dangle-foot types were also represented. The cases had been operated from two to ten years previously. The ages of the patients varied from three to twenty years, with an average age of eight to ten years.

In all cases of calcaneovalgus, the peronei were transplanted into the Achilles, with the result that function was increased. In 12 cases in which varus deformity predominated originally, the peronei were fixed to the fibula, with satisfactory results.

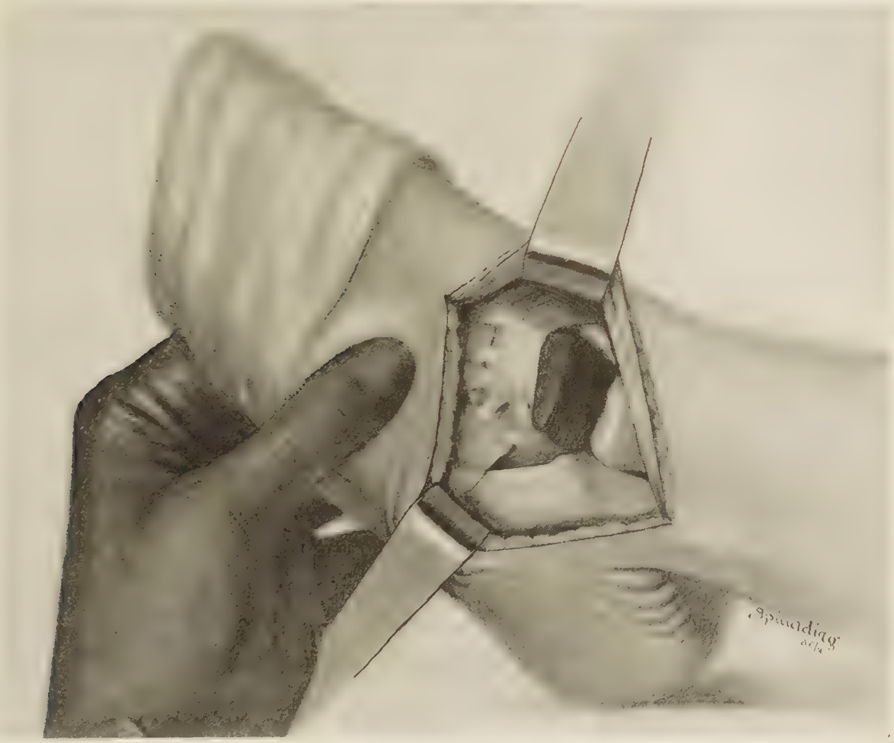

Fig. 99.—*Astragalectomy. The Whitman operation.* Fibula and tibia being inserted into pockets.

Out of the 247 cases examined, there were 12 in which postoperative deformity had developed following operation. Seven of these cases had been of the varus type; 1 patient had refused after-treatment, 1 had insisted that the family physician change the plaster bandage, and the remaining 5 had developed deformities from improper after-care in our clinics. These deformities had been corrected before the patient was finally discharged.

One case of equinovarus deformity had recurred. The patient had not returned to the clinic for after-care.

There was 1 case of sloughing in a toe from swelling, which developed because the leg had not been elevated when the patient was treated at home. The end-result was good.

Case XIII.—E. D., aged eight years. Calcaneovalgus deformity.

Complaint: Deformity of the left foot.

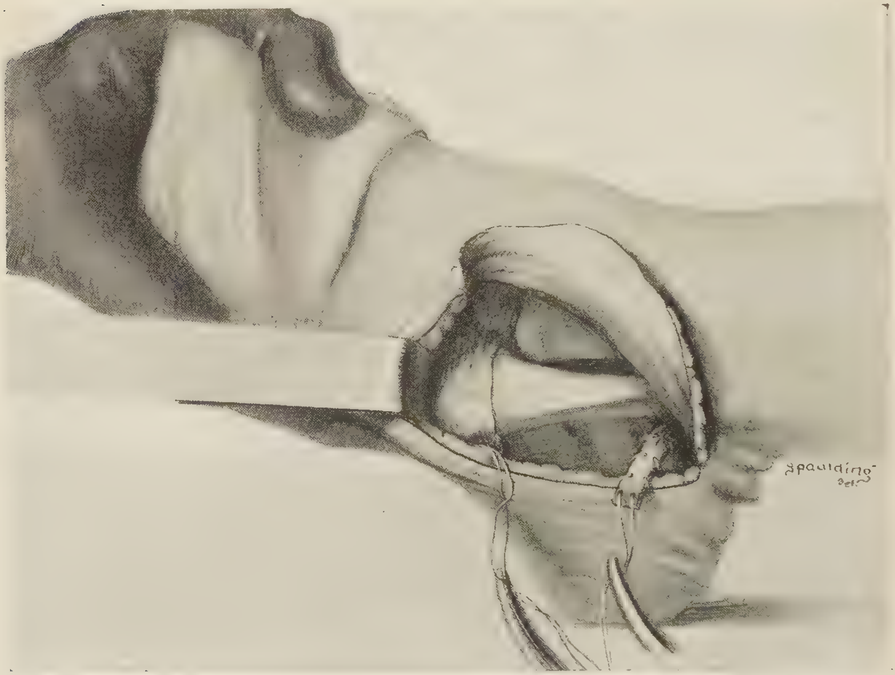

Fig. 100.—*Astragalectomy. The Whitman operation.* Backward displacement of the foot. Note that the fibula is carried well forward.

Duration: Four years.

Cause: Infantile paralysis.

Physical Examination: There were no contractures. The gastrocnemius and plantar flexors were absent. The quadriceps, anterior tibial, and peroneal muscles were strong. There was shortening amounting to $1\frac{1}{2}$ inches. The foot was in calcaneovalgus deformity (Fig. 108).

Treatment: December, 1914. An astragalectomy with transplantation of the peroneal tendons into the tendo Achillis was performed.

Result: January 22, 1924. The foot was at a right angle in excellent posterior displacement and in good alignment (Fig. 109). There was 10 degrees motion in the mid-tarsal joint. The patient was wearing a lift under the heel and sole of the shoe to compensate for shortening.

Case XIV.—J. B. Calcaneovalgus deformity.
Complaint: Deformity of the right foot.
Cause: Infantile paralysis.

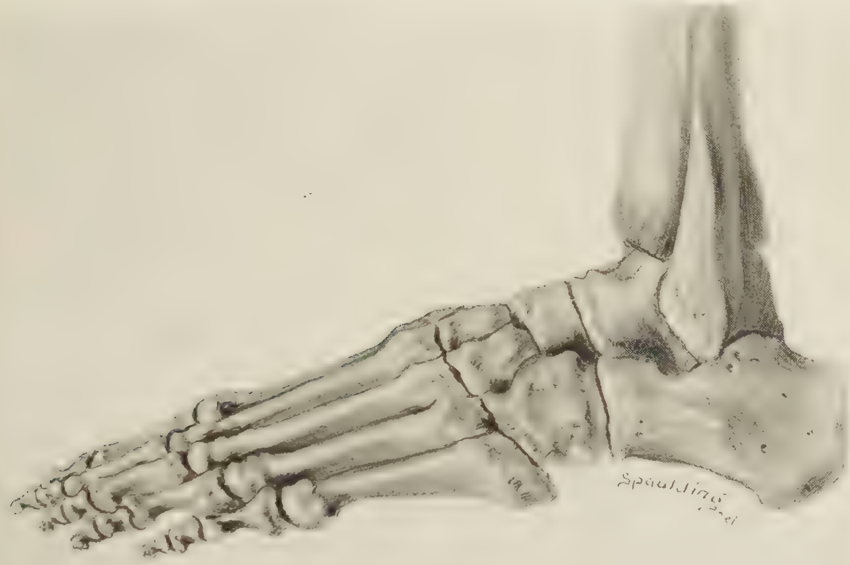

Fig. 101.—*Astragalectomy. The Whitman operation.* Normal relation of the tarsal bones.

Physical Examination: The outer dorsiflexors of the leg and the plantar flexors of the toes had good power. The gastrocnemius and the muscles on the inner side of the foot were absent.

Treatment: April 17, 1917, an astragalectomy and transplantation of the peroneal tendons into the Achilles tendon were performed.

Result: October 2, 1917. The foot was in excellent position and had 15 to 20 degrees motion (Fig. 110). The patient was wearing a $\frac{5}{8}$ inch lift on the heel of the shoe to compensate for shortening.

Case XV.—L. M., aged fourteen years.
Complaint: Walks with heel-limp.

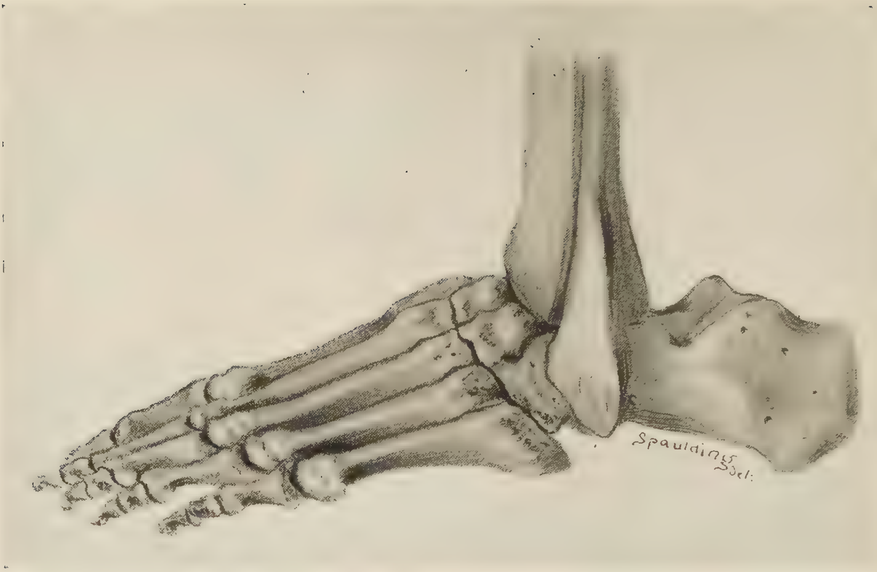

Fig. 102.—*Astragalectomy. The Whitman operation.* Relations following an astragalectomy properly done.

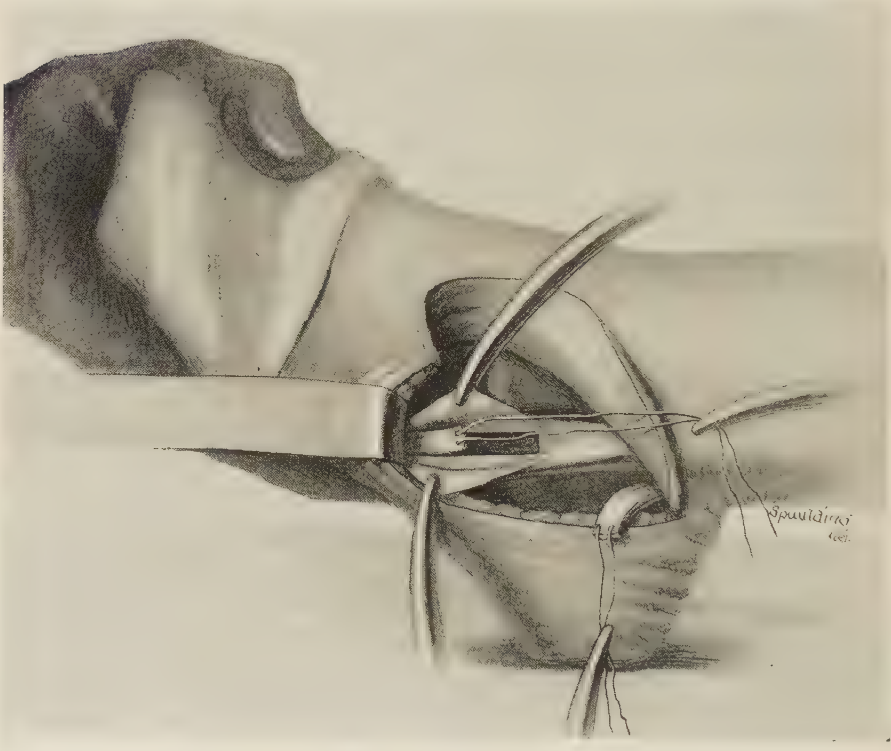

Fig. 103.—*Astragalectomy. The Whitman operation.* Fixation of the distal ends of the peroneal tendons into the fibula.

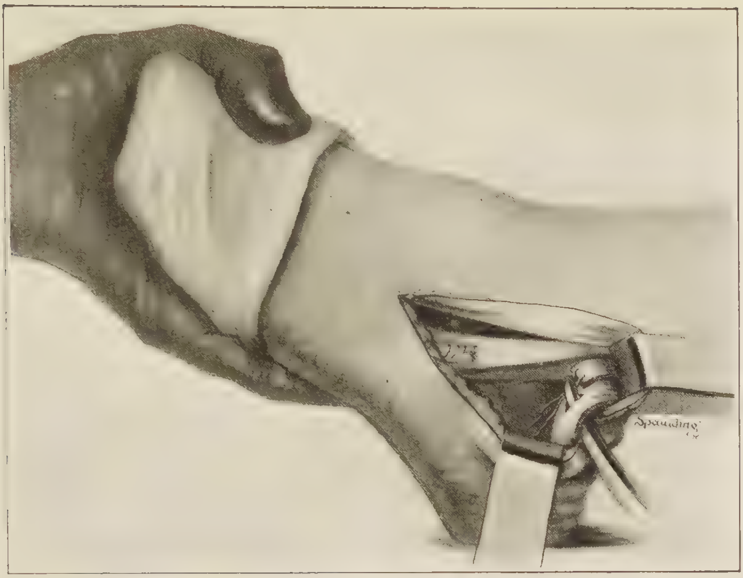

Fig. 104.—*Astragalectomy. The Whitman operation.* Disposition of the proximal ends of the peroneal tendons into the Achilles tendon. The distal ends have been fixed into the fibula.

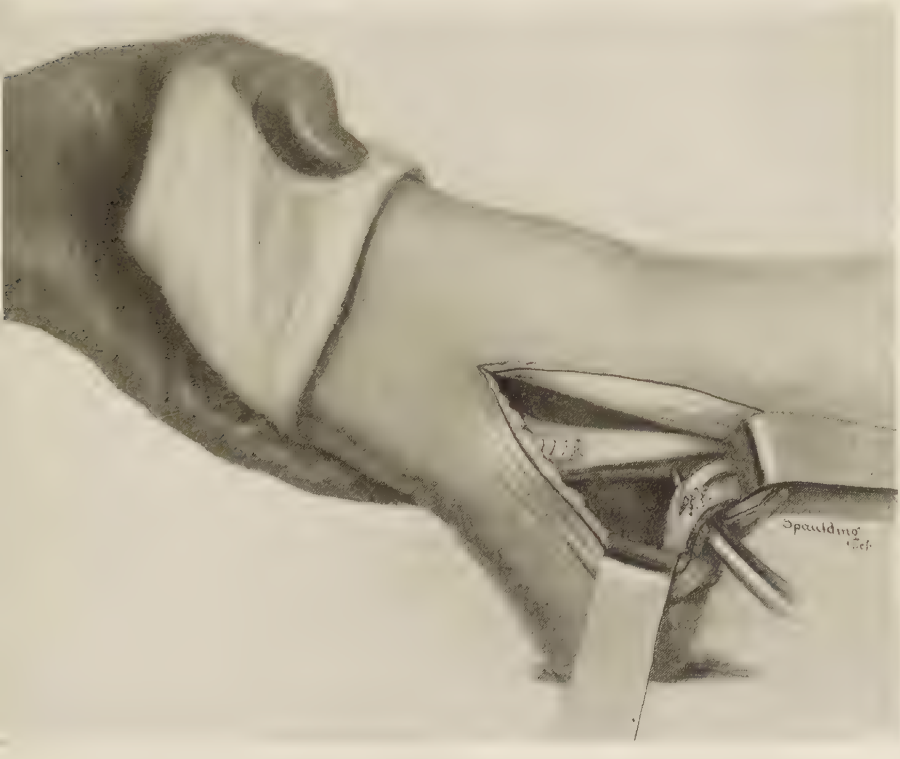

Fig. 105.—*Astragalectomy. The Whitman operation.* Suture of the proximal ends of the peroneal tendons into the Achilles tendon. The distal ends have been fixed into the fibula.

Duration: About twelve years.

Cause: Infantile paralysis.

Physical Examination: August 7, 1923. The left foot was in calcaneus deformity and slight cavus. The leg, especially the calf, was atrophied. The iliopsoas, quadriceps, and tibialis anticus were strong. The gastrocnemius and the peronei had no power (Fig. 111).

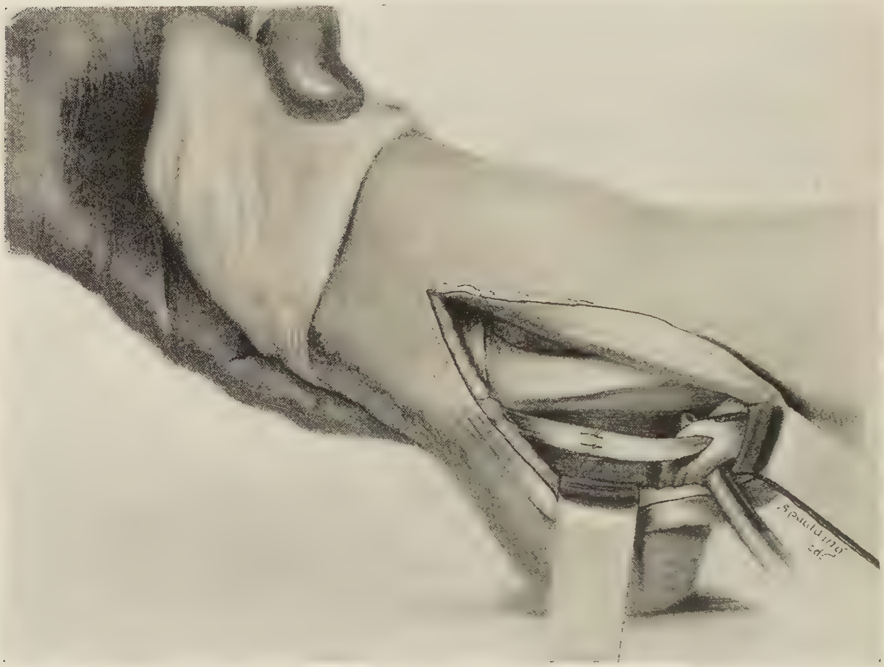

FIG. 106.—*Astragalectomy. The Whitman operation.* Disposition of the peroneal tendons into the Achilles tendon, and resuture to their old stumps.

Treatment: October 17, 1923. Astragalectomy and a tendon fixation of the flexor longus hallucis to prevent plantar flexion deformity of the great toe were performed.

Result: January 22, 1924, three months after operation, the patient could bear weight and was to begin walking (Figs. 111, 112, and 113).

Result: January, 1926. The foot was in excellent position, and function was perfect.

Arthrodesis.—The object of an arthrodesis is to stiffen a joint which is not under muscle control. Such a process, by restoring stability to the foot, increases its function.

Objections may be made to stiffening a joint. However, a patient can walk better with a foot in proper position, even though one or more joints are stiff, than he can with a foot that is insufficiently controlled by muscles, or that is handicapped by braces. Following arthrodesis, the foot is stable and functional, and it is not necessary to wear apparatus. After operation many patients do not limp at all, and others only slightly.

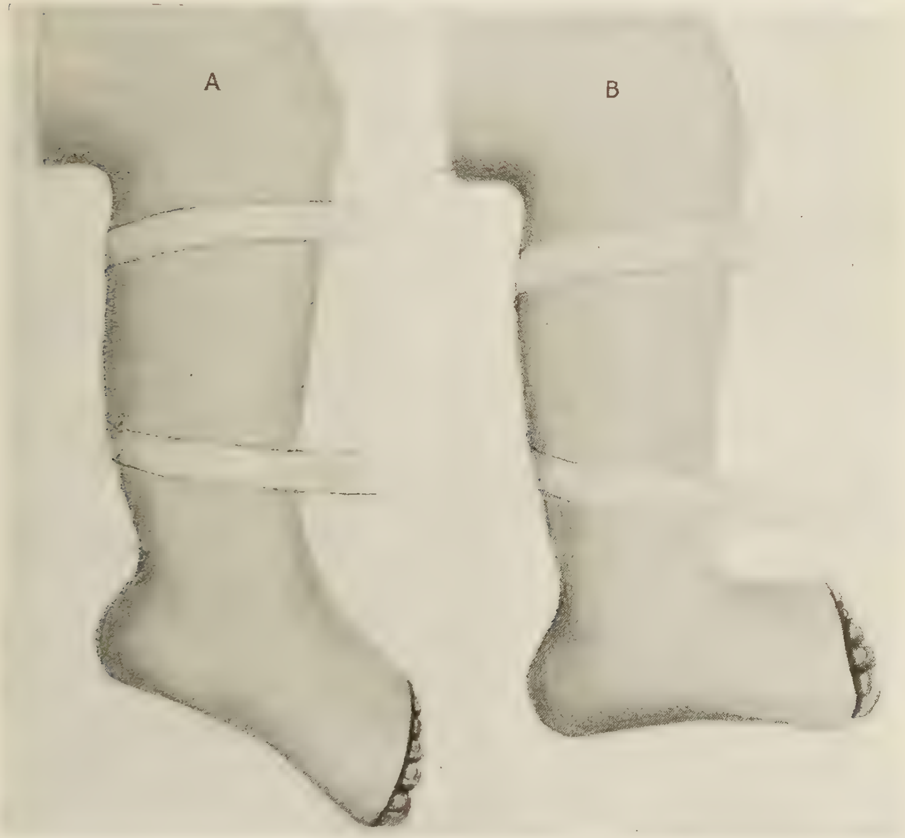

FIG. 107.—*Astragalectomy. The Whitman operation.* In plaster following operation. The foot is in slight equinovalgus and the knee is flexed.

An arthrodesis should not be performed until two years have elapsed from the onset of the disease, and not before the child has reached the age when the bones are sufficiently ossified to ensure ankylosis. The best results are obtained in cases over sixteen years of age. Some years ago, the writer examined a group of children between the ages of eight and twelve years, upon whom various types of arthrodeses had been performed. The joints had not stiffened, and deformity had recurred; no traces of bony union were visible in radiograms. These children were too young to have been subjected to this operation.

The selection of the joints to be stiffened depends upon the muscle power remaining in the foot and upon the age of the patient. Motion in the foot takes place at the ankle, at the subastragalar, and at the mid-tarsal joints. If possible, the motion in the ankle-joint must be preserved, and it is only in exceptional cases that this joint is sacrificed. In the majority of cases, stability may be restored satisfactorily by stiffening the astragalo-scaphoid joint or subastragalar joints.

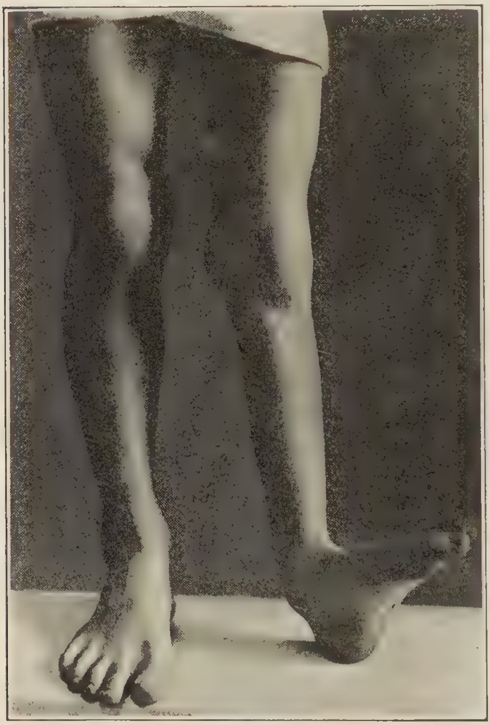

Fig. 108.—Case XIII. E. D. *Calcaneovalgus deformity.* Anterior view before astragalectomy.

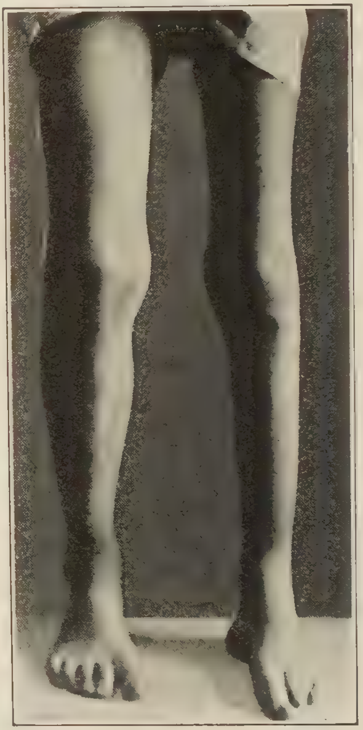

Fig. 109.—Case XIII. E. D. *Calcaneovglaus deformity.* Anterior view ten years after astragalectomy.

Astragalo-scaphoid Arthrodesis.—This operation may be used to control valgus deformity in young children, when conservative

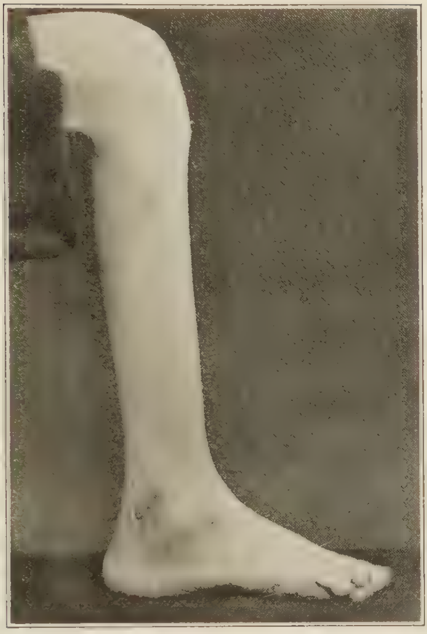

Fig. 110.—Case XIV. J. B. *Calcaneovalgus deformity.* Lateral view six months after astragalectomy.

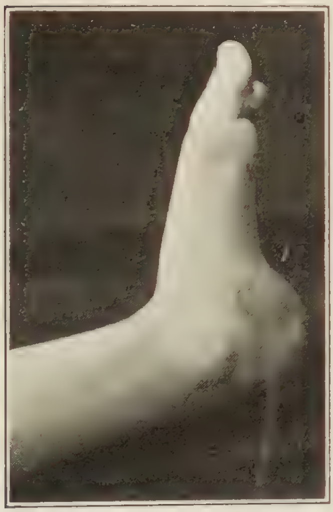

Fig. 111.—Case XV. L. M. *Calcaneocavus deformity.* Lateral view before astragalectomy.

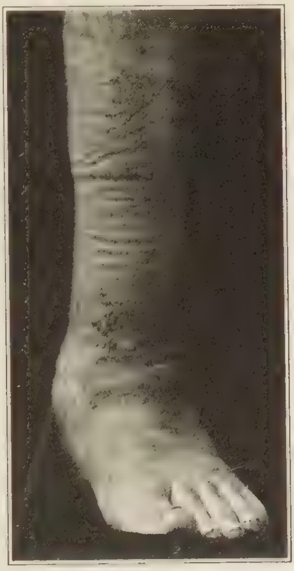

Fig. 112.—Case XV. L. M. *Calcaneocavus deformity.* Anterior view three months after astragalectomy.

means have failed. It may be performed in children under twelve years of age, and before the two-year period has elapsed from the onset of the attack. In such cases the result may be a fibrous ankylosis, which affords only temporary relief, but the operation has the advantage of holding the deformity in check until further stabilizing operations may be carried out.

An astragalo-scaphoid arthrodesis may be performed to restore the balance of the foot in children over twelve years of age, when the muscle groups other than the tibiales are strong.

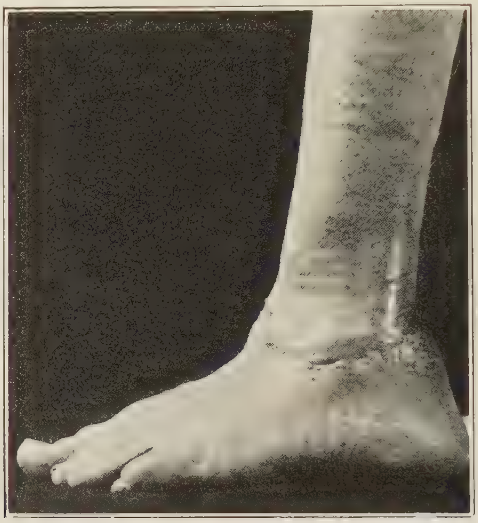

FIG. 113.—Case XV. L. M. *Calcaneocavus deformity.* Lateral view three months after astragalectomy.

When arthrodesis does not seem sufficient to overcome the valgus deformity, the operation may be supplemented by a transplantation of the peronei into the scaphoid, or by subperiosteal fixation of the anterior tibial tendon into the tibia.

When used in carefully selected cases, the results of astragalo-scaphoid arthrodeses are satisfactory. Their success depends a great deal upon the careful execution of the operative technique.

Operative Technique.—The leg from the toes to the knee is prepared in the usual way.

A curved incision is made over the lateral aspect of the astragalo-scaphoid joint, the flap pointing downward (Fig. 114). The skin and fat are retracted backward, and the joint capsule exposed (Fig. 115). The astragalo-scaphoid joint is opened, and the foot

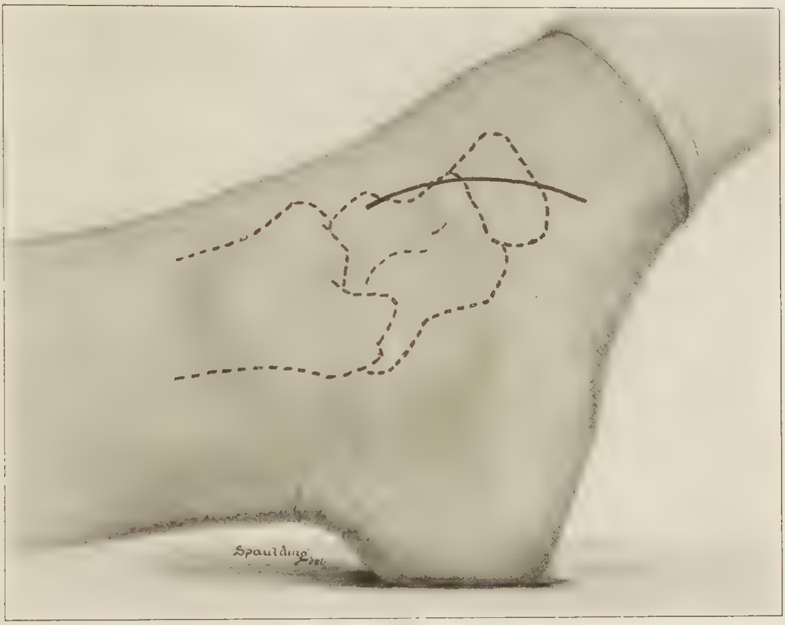

FIG. 114.—*Astragalo-scaphoid arthrodesis.* Incision over astragalo-scaphoid joint.

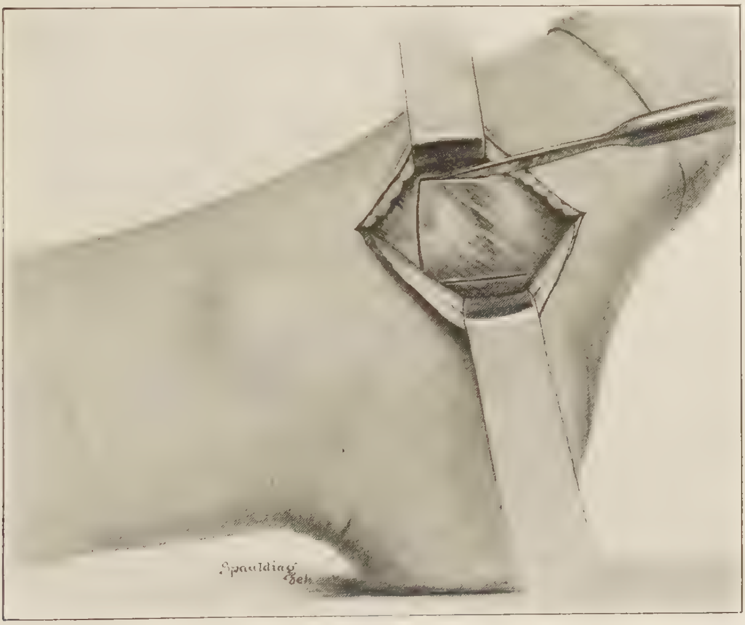

FIG. 115.—*Astragalo-scaphoid arthrodesis.* Skin and fat retracted backward, exposing joint capsule.

everted to expose as much of the head of the astragalus as possible. The cartilage is removed from the head of the astragalus and from the opposing surface of the scaphoid (Fig. 116). The foot is forcibly manipulated into varus. The periosteum and capsule are sutured tightly with interrupted chromic gut, and the skin is closed with continuous catgut. A plaster bandage is applied from the toes to the knee, with the foot in the position of marked varus and slightly above a right angle.

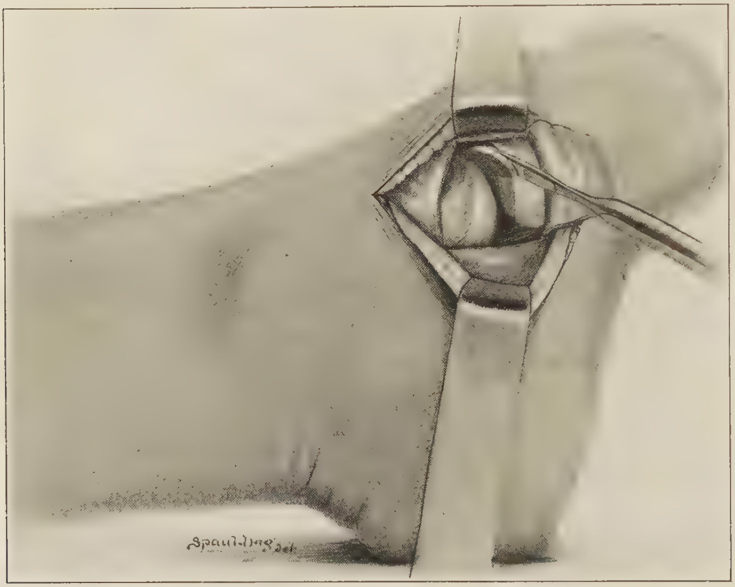

Fig. 116.—*Astragalo-scaphoid arthrodesis.* Removal of cartilage from the head of the astragalus and from the scaphoid.

The plaster is worn for ten weeks. Weight-bearing is allowed after the sensitiveness has disappeared, usually in about ten days. After the removal of the bandage, a Whitman brace and Thomas heel should be worn to prevent strain and to hold the foot well overcorrected for six months.

Case XVI.—L. B., aged eight years.
Complaint: Paralysis of both legs.
Duration: Old-standing.
Cause: Infantile paralysis.
Physical Examination: The child stood with marked hyperextension of the right knee and marked valgus of both feet (Fig. 117).

Treatment: September 4, 1925, an astragalo-scaphoid arthrodesis of the right foot was performed. Thus far, this is the only correction which has been made.

Result: March, 1926. The foot is in good position, and function is definitely improved (Fig. 118).

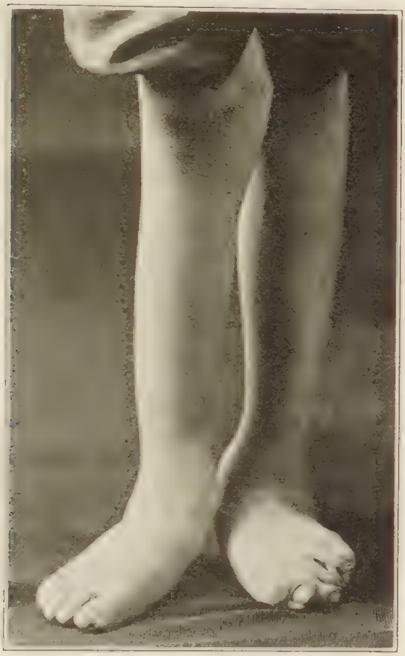

Fig. 117

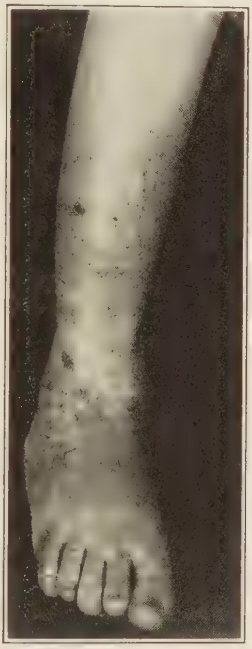

Fig. 118

Fig. 117.—Case XVI. L. B. *Valgus deformity of both feet.* Antero-posterior view before astragalo-scaphoid arthrodesis, showing marked valgus of both feet.

Fig. 118.—Case XVI. L. B. *Valgus deformity of both feet.* Antero-posterior view of the right foot, six months after astragalo-scaphoid arthrodesis.

Subastragalar Arthrodesis.—While astraglectomy is a most satisfactory stabilizing operation for many paralytic conditions, it is likely to result in a painful foot if performed in children more than fifteen years of age. There are also cases between the ages of twelve and fifteen years in which it is more advisable to stiffen the joints below the astragalus than to perform an astragalectomy. The surgeon must determine which operation is preferable.

Indications and Contraindications.—Subastragalar arthrodesis may be used in severe valgus deformity associated with a general muscle involvement, if the age of the patient precludes the use of astragalectomy. When used in talipes equinovalgus, a subastragalar arthrodesis primarily controls the valgus deformity, and the equinus element is indirectly compensated by the overcoming of the tarsal eversion and by the posterior displacement of the foot. A subastragalar arthrodesis also may be used in valgus deformity associated with calcaneus, in calcaneovarus, in varus, equinovarus, and flail-foot, when the patient is between the ages of twelve and twenty years. If any cavus is present, a V-shaped wedge is removed from the mid-tarsal region to correct this deformity.

Subastragalar arthrodesis is not indicated in talipes equinus.

Operative Technique.—The technique of this operation has been worked out by several surgeons. The Davis[24] and Hoke[25] operations are those most generally known. The writer will report the technique of these operators and then cite his own method of arthrodesis.

Davis Operation.—Two small lateral incisions are made; one on the outer side of the foot opposite the tip of the external malleolus to expose the posterior portion of the astragalo-calcanean joint, and the other on the inner side of the foot about $\frac{3}{4}$ inch below and in front of the internal malleolus, to expose the adjacent surfaces of the scaphoid and the astragalus as well as the sustentaculum tali. With a gouge $\frac{1}{4}$ inch wide, the line of the joint right across below the external malleolus is dug up and made as rough as possible. The gouge is withdrawn, inclined anteriorly, and the outer portion of the head of the astragalus and the adjacent articular surface of the scaphoid are dug up. On the inner side of the foot, after the posterior tibial tendon has been hooked out of the way to avoid cutting it, the chisel is pushed in $\frac{3}{4}$ inch below the internal malleolus, and the surface of the subastragalar joint between the sustentaculum tali and the astragalus is dug up. Then the adjacent surfaces of the astragalus and scaphoid are dug up. The foot is left in this disturbed condition. The incisions are closed. The foot is held in plaster for two or three months until union is firm.

Comment.—Although this operation is based upon sound principles, it is a mutilating one and not a clear surgical measure. A firm ankylosis does not always result. The operation also does not provide for the establishment of a definite displacement of the foot.

Hoke Operation.—This operation consists of stabilizing the subastragalar and astragalo-scaphoid joints and correcting the posterior foot deformity at the same time.

Many successful results have been reported. In a series of 104 cases operated by Hoke, 57 cases, which he had the opportunity to examine six months to three years after operation, had stable feet. In the cases of foot-drop in which the operation was used, the toes have always cleared the ground without a high knee action, except when the quadriceps extensor was weak, the hamstrings and triceps suræ strong, and the anterior group below the knee totally paralyzed.

Operative Technique.—The skin incision extends from over the external portion of the head of the astragalus downward and backward to the peroneal tendons below the end of the fibula. The adipose tissue of the "subastragaloid fossa," that is, the space bounded above by the neck of the astragalus, below by the superior surface of the os calcis, and behind by the body of the astragalus, is split. Its upper and lower portions are dissected out, leaving the under surface of the neck and the adjacent portion of the body of the astragalus and the superior surface of the os calcis bare. With a knife the superior surface of the neck of the astragalus is denuded of tissue. The head of the astragalus is freed from the scaphoid, by first cutting the astragalo-scaphoid ligament, then beginning low and externally and sweeping around the head, ending internally. A portion of the inferior surface of the body of the astragalus and a portion of the adjacent surface of the os calcis are removed. The neck of the astragalus is cut through where it joins the body. The head of the astragalus is levered partly out, and the remaining attachments are cut with scissors. The neck and head of the astragalus are placed in a towel. The scaphoid and the facet on the superior surface of the os calcis are denuded of cartilage. The posterior foot deformity may now be corrected as required; the posterior end of the os calcis may be set in line with the center of the leg, the os calcis may be shifted laterally in line with the central axis of the leg, lateral rotation of the os calcis may be corrected, and the foot may be shifted backward.

The head of the astragalus is denuded of cartilage.

The position in which the astragalus is replaced depends upon the type of paralytic foot; it is put back in a different position for drop-foot, club-foot, and flat-foot. In simple drop-foot, for instance, the astragalus is too long to go back in its place when the anterior foot is cocked up. The astragalus is held on a wooden block, and with a hammer and osteotome, just enough of the posterior end is removed so that the bone piece sinks in neatly between the scaphoid and the body of the astragalus without hindering the cocking up of the anterior foot.

After the astragalus has been replaced, the foot is brought into slight dorsiflexion. The assistant sews the subcutaneous tissues with interrupted No. 1 catgut sutures, and the skin with interrupted silk sutures. A plaster bandage is applied from the toes to the knee.

If it is not possible to correct the downward pitching of the shafts of the metatarsals to the degree desired, a manipulation may be done, the foot being forced downward and the anterior ends of the metatarsals upward.

In the case of children, the cast is worn from six and a half to seven and a half weeks, and in the case of adults from eight to nine weeks. After-treatment consists simply of two weeks' massage and the wearing of a proper shoe.

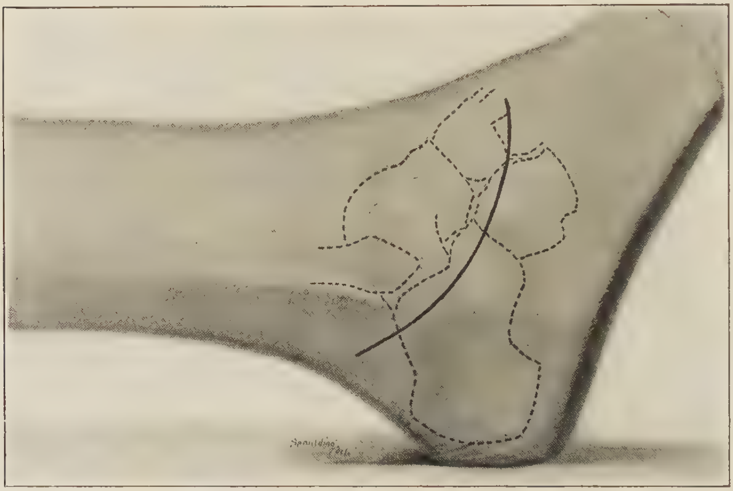

FIG. 119.—*Subastragalar arthrodesis.* Incision around the external malleolus.

Comment.—Hoke's operation undoubtedly secures arthrodesis in the subastragalar joints, but it is unnecessarily complicated. The removal of part of the neck of the astragalus and its replacement are difficult and attended with risks. Also, good backward displacement of the foot, which is essential to the establishment of a new weight-bearing line, cannot be obtained by this operation.

Subastragalar Arthrodesis (MacAusland's Method).—By means of this technique, it is possible to obtain a good surgical exposure and to displace the foot to any degree desired.

Operative Technique.—A curved or L-shaped incision is made around the external malleolus, extending forward over the head of the astragalus. This incision is similar to that made in an astragalectomy, except that it is carried a little farther forward. In order to avoid cutting the ligaments between the astragalus and the fibula, it is well to make the incision low enough to pass over the joint between the astragalus and the os calcis, and then to carry it forward to the head of the astragalus (Fig. 119).

The sole of the foot is inverted, without disturbing the ligamentous attachments of the tibia, fibula, and astragalus. A transverse section is made from the tip of the os calcis through the top of the tarsal region (Fig. 120). A parallel transverse section is made in the lower third of the astragalus. The intervening bone structure is removed (Fig. 121). The lower part of the foot now can be displaced posteriorly to any degree (Fig. 122). The bulging surface of the anterior part of the astragalus is then removed.

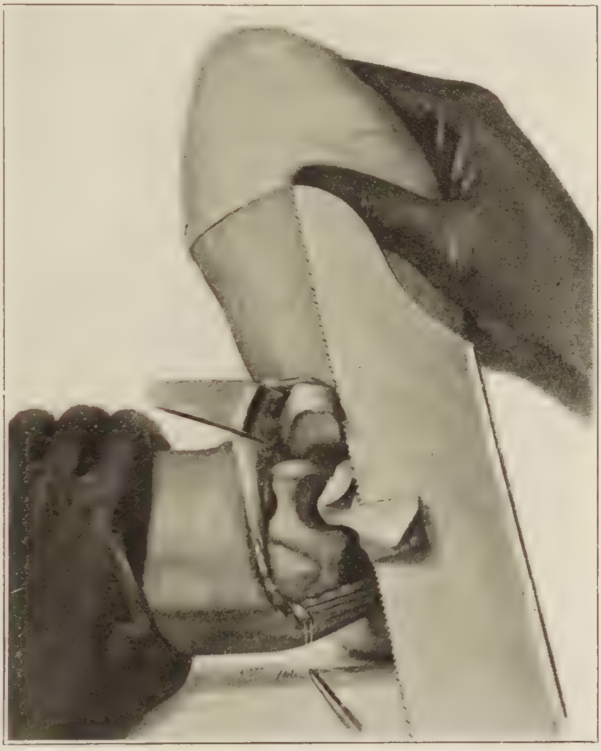

FIG. 120.—*Subastragalar arthrodesis.* The removal of a transverse section from the tip of the os calcis through the top of the tarsal region.

Any cavus deformity may be overcome by the removal of a V-shaped wedge, whose apex is toward the plantar surface and whose base is toward the astragalus.

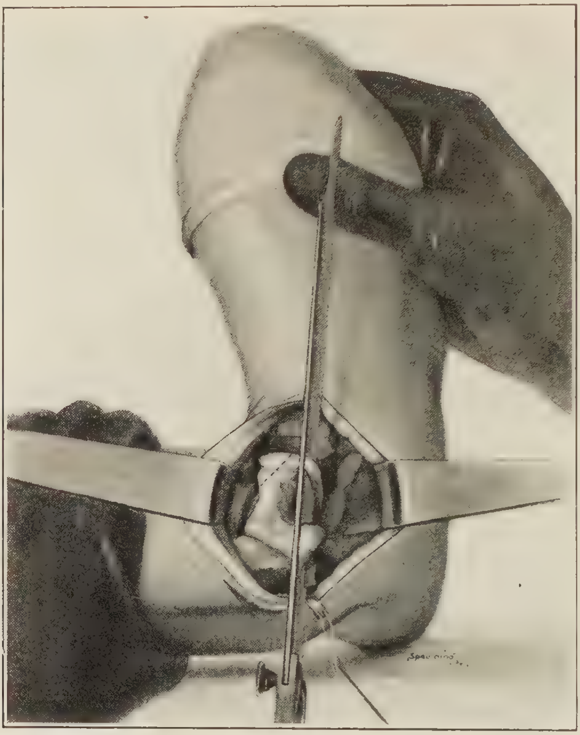

FIG. 121.—*Subastragalar arthrodesis.* The removal of a transverse section from the lower third of the astragalus.

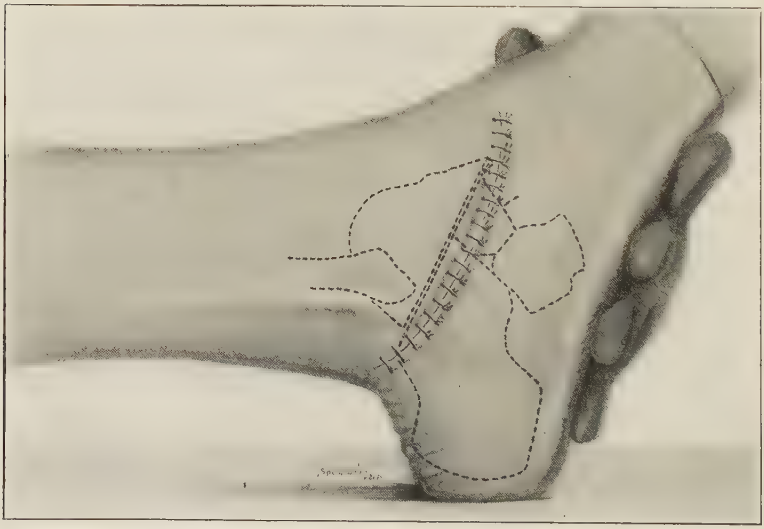

FIG. 122.—*Subastragalar arthrodesis.* Posterior displacement of the tarsus on the astragalus.

The above technique has been carried out in 6 cases, and very satisfactory results obtained. In 3 cases, a subastragalar arthrodesis alone was performed. In the other 3 cases, in addition to the arthrodesis, a V-shaped wedge was removed from the mid-tarsal joint to correct the cavus deformity.

Case XVII.—M. K., aged four years.

Complaint: Paralysis and deformity of both feet.

Duration: Two years.

Cause: Infantile paralysis.

Physical Examination: In the right leg, the iliopsoas and quadriceps had good power; the anterior and posterior tibial muscles were without power. The patient stood with the foot in marked valgus and pronation. In the left leg, the posterior tibial muscle was present, but weak. The anterior tibial muscle was absent. The foot was in moderate pronation. Both feet showed some toe-drop.

Treatment: The previous treatment had consisted of massage and muscle training. The patient had also worn Thomas heels.

March 14, 1919. A double astragalo-scaphoid arthrodesis was performed, and splendid overcorrection obtained. One year after operation, the patient walked with the ankles in good position. At this time, Whitman braces were worn as well as a Thomas heel and sole on the right foot. During the next year, the Achilles tendons of both feet showed a tendency to contract.

July 27, 1920. Both tendons were taut, allowing motion to only a right angle. The right foot was in slight valgus. Except for the contraction of the Achilles tendon, the left foot was in good position.

September 29, 1920. Tenotomies of both Achilles tendons were performed. Two months later, both feet were straight, although the right foot still showed a tendency toward valgus.

September 30, 1921. One year later the right foot had relapsed, and a second astragalo-scaphoid arthrodesis with a Gallie fixation of the anterior tibial muscle into the tibia was performed. From this time on, the patient held her feet in good position and walked better.

June 20, 1923. Both arches were perfectly formed. The left foot was in excellent position. The right foot was straight, but there was a slight tendency to pronation. The peronei and outer dorsal flexors in this foot were unusually strong. Although it was thought that the patient could voluntarily overcome the pronation, this was found impossible. By *March 1, 1924*, she was walking with both feet pronated, especially the right. There was considerable eversion of both legs. The patient had about 10 degrees to 15 degrees knock-knee, and in walking, the knees interfered, a factor which contributed much to the outward rotation.

The condition grew worse. By *April 30, 1924*, the patient walked with considerable valgus. The mid-tarsal joints, however, were holding firmly. In both feet, all muscles except the anterior tibials had power.

June 18, 1924. A double subastragalar arthrodesis was performed.

Result: October 10, 1924. The feet were in excellent position, and the patient walked well. The condition continued to improve. (Fig. 123) shows the result eight months after operation.

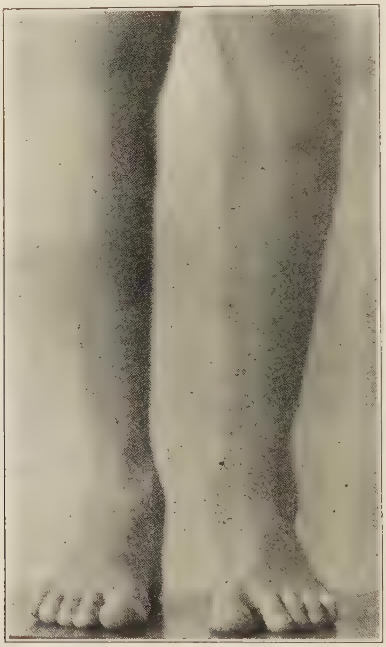

FIG. 123.—Case XVII. M. K. *Valgus deformity of both feet.* Antero-posterior view, eight months after subastragalar arthrodesis of both feet.

CASE XVIII.—M. H., aged three years.

Complaint: Lameness of left foot.

Duration: One year.

Cause: Infantile paralysis.

Physical Examination: All muscles of the left side, except the gastrocnemius, had power (Fig. 124).

Treatment: For about eight weeks, the child had worn a shoe with an upright and a reverse stop-joint, but power had not returned in the gastrocnemius.

September 30, 1924. A subastragalar arthrodesis and a tenotomy of the plantar fascia were performed.

Result: December 23, 1924. A good displacement had been obtained. The shortening was compensated by a raise on heel and sole.

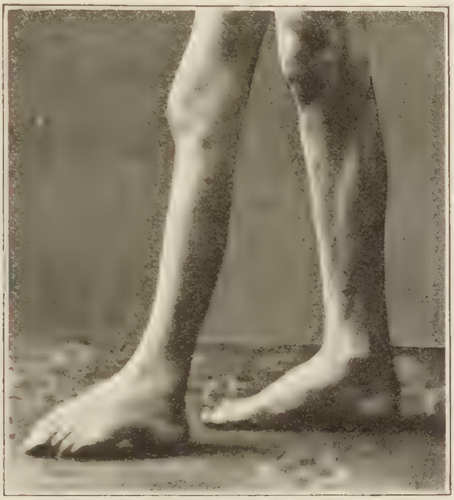

FIG. 124.—Case XVIII. M. H. *Calcaneocavus deformity of the left foot.* Lateral view before operation.

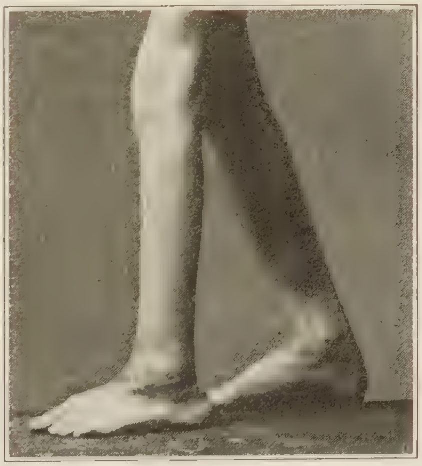

FIG. 125.—Case XVIII. M. H. *Calcaneocavus deformity of the left foot.* Correction, one year and seven months following subastragalar arthrodesis.

(Fig. 125) shows the position of the foot, one year and seven months after operation.

CASE XIX.—T. L., aged nine years.

Complaint: Paralysis of the right foot.

Duration: Seven years.

Cause: Infantile paralysis.

At the age of two years, this patient suffered an attack of infantile paralysis which affected practically the entire right side. The greatest residual weakness was located in the right leg and foot, and the patient walked with a limp.

Physical Examination: The right thigh was somewhat weak. The leg muscles, with the exception of the gastrocnemius and posterior tibial, had some power. There was some cavus deformity (Fig. 126).

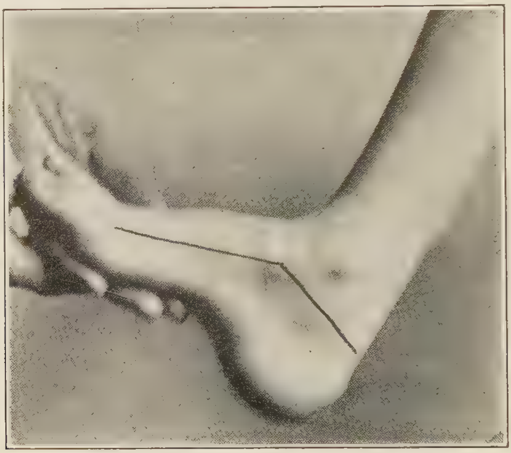

Fig. 126.—Case XIX. T. L. *Calcaneocavus deformity.* Lateral view before operation.

Treatment: July 27, 1922. The Achilles tendon was shortened by open operation, and a plaster bandage applied with the foot in plantar flexion. The plaster was worn for twelve weeks, then an inner upright equipped with a reversed stop-joint permitting plantar flexion to 7 degrees was applied to the shoe. Massage was given for three weeks after the removal of the plaster.

March 24, 1923. There was some power in the gastrocnemius, and the contour of the foot was better. The brace was discarded, and the foot strapped in the position of slight plantar flexion.

During the next few weeks a definite spastic condition developed. The child walked with the foot in valgus deformity. The gait was shuffling, due probably to the mild meningeal infection.

March 14, 1924. The patient still walked with the foot in valgus, and the os calcis was tilted upward. The Achilles tendon was not so lax as originally. The peronei were definitely spastic. Spiral strapping was applied to hold the foot in varus. The foot was restrapped several times at intervals of two weeks, but without relief.

July 9, 1924. A subastragalar arthrodesis was performed according to the writer's technique.

Result: October 4, 1924. The plaster bandage was removed. The foot was in excellent position. A shoe to which was attached an outer upright equipped with a right-angle stop-joint was advised.

August 1, 1925, nearly one year after operation, the patient was walking well without a brace. The foot was in excellent position (Fig. 127).

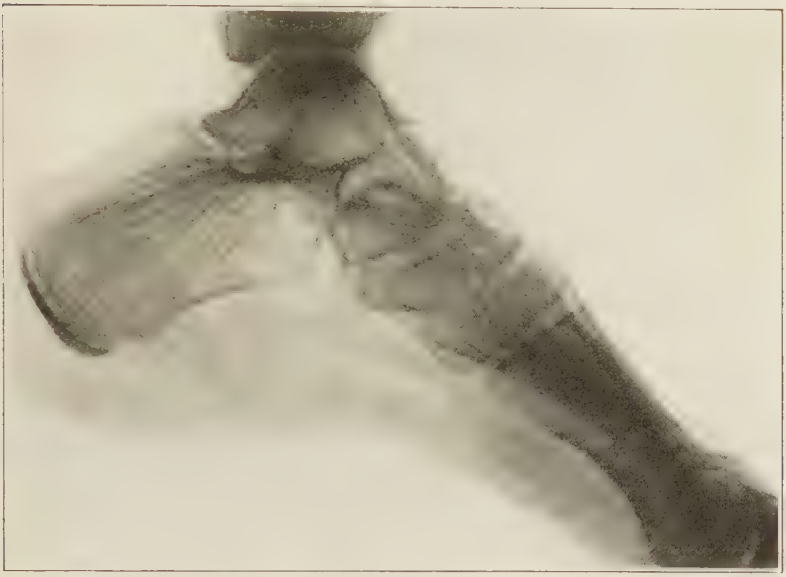

FIG. 127.—Case XIX. T. L. *Calcaneocavus deformity.* Radiogram showing correction one year after subastragalar arthrodesis.

CASE XX.—H. R., aged twenty-three years. Calcaneocavus deformity (Fig. 128).

Complaint: Lameness.

Duration: Twenty-one and a half years.

Cause: Infantile paralysis.

Treatment: January 14, 1925. The writer's subastragalar arthrodesis was performed, and a V-shaped wedge taken out of the midtarsal region.

Result: February 9, 1925. The foot was in excellent displacement. There were signs of union in the subastragalar joints.

March 9, 1925. The plaster bandage was removed, and an outer upright with inner T-strap applied to the shoe.

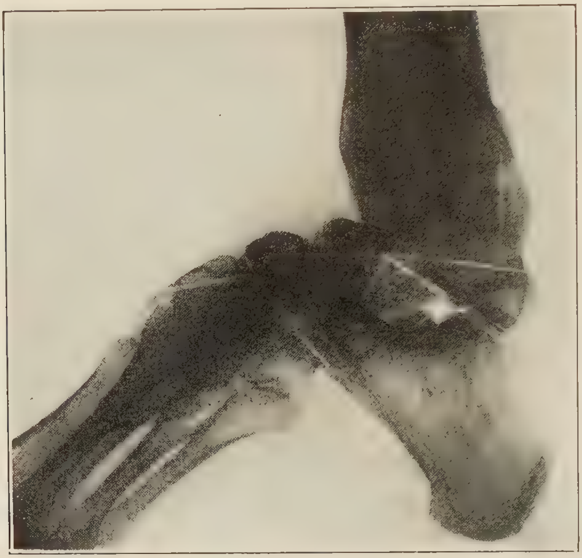

Fig. 128.—Case XX. H. R. *Calcaneocavus deformity.* Lateral radiogram before subastragalar arthrodesis.

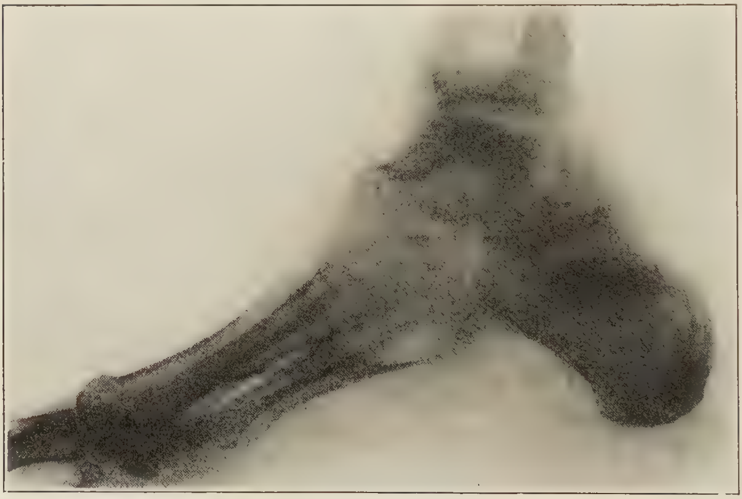

Fig. 129.—Case XX. H. R. *Calcaneocavus deformity.* Lateral radiogram seven months after subastragalar arthrodesis.

August 24, 1925. The radiogram and photograph (Figs. 129 and 130), taken seven months after operation, show an excellent result.

Case XXI.—D. B., aged ten years.

Complaint: Walks with limp of the right leg.

Duration: Of long-standing.

Cause: Infantile paralysis.

Physical Examination: October 16, 1923. The patient stood with a total left scoliosis. The left leg was about ¼ inch shorter than the right and was blue and mottled in its lower half. All the muscles of the foot, except the gastrocnemius, had slight power. The forefoot was broad, and the plantar fascia tense and hard. The foot was in the deformity of cavus.

Treatment: October, 1923. A tenotomy of the plantar fascia was done. Then, for more than two years, the patient wore a boot with a built-up heel and sole, proper adjustment being made as required.

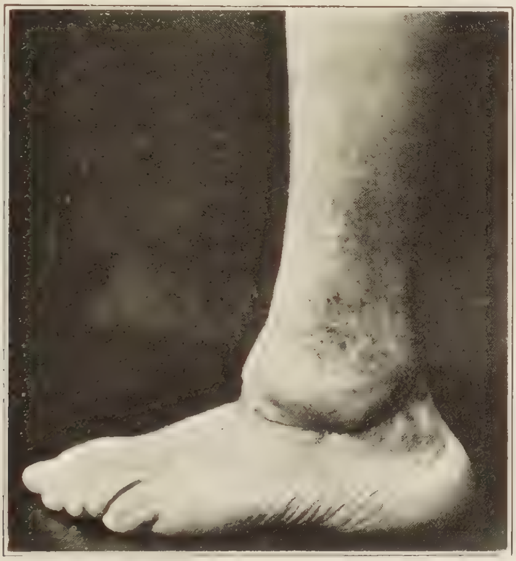

Fig. 130.—Case XX. H. R. *Calcaneocavus deformity.* Lateral view seven months after subastragalar arthrodesis.

April 28, 1925. Physical examination showed no return of power in the gastrocnemius. The foot was painful, and the cavus seemed to be increasing. There was about 80 degrees deformity of the forefoot. A tenotomy of the plantar fascia and stretching were done. A plaster bandage, reënforced on the sole, was applied as far up as the knee. Weight-bearing was begun in three days.

June 23, 1925. The regular subastragalar arthrodesis with removal of a plantar V-shaped wedge was done. A plaster bandage was applied, the lower part of which was to be worn for ten weeks, and the upper part removed at the end of ten days. Walking was allowed in four weeks (Fig. 131).

Result: August 18, 1925. The foot was in excellent position. There was good motion in the ankle-joint. Photographs (Figs. 132, 133, and 134) taken *September 29, 1925,* and radiogram (Fig. 135) taken eight months after operation, show the excellent position of the foot.

Arthrodesis of the Ankle-joint.—This operation is indicated in flail-feet in which no muscle power remains, or in adult cases of severe muscle weakness. Arthrodesis of the ankle now is performed less often than formerly, as operations that afford better functional results have been devised for paralytic feet in which any slight muscle power remains. Occasionally, however, the case is seen in which sacrifice of the ankle-joint is the only means by which the foot may be made stable.

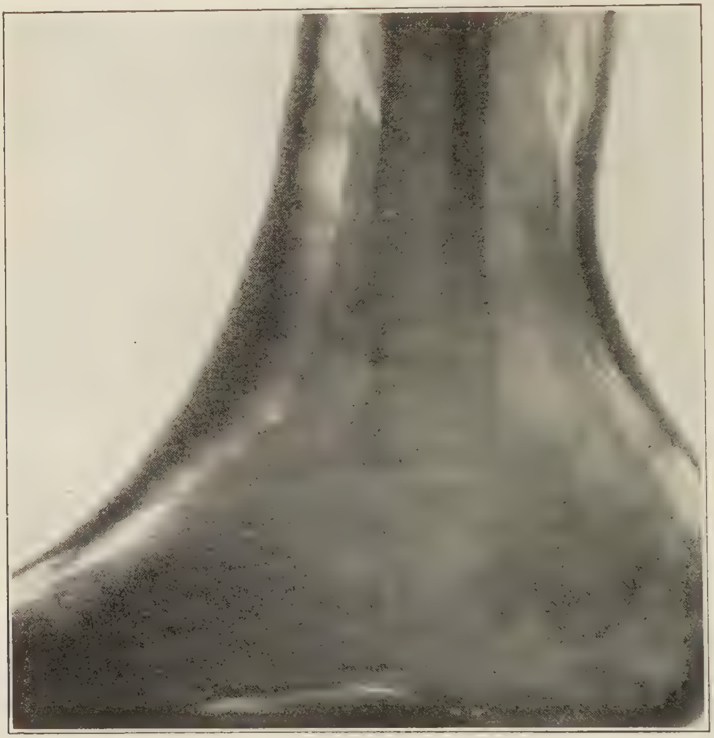

FIG. 131.—Case XXI. D. B. *Cavus deformity.* Radiogram showing foot in plaster bandage three weeks after subastragalar arthrodesis.

Operative Technique.—The leg, from the toes to the knee, is prepared in the usual aseptic manner. A tourniquet is used in operating.

A semicircular skin incision is made, beginning at a point between the prominence of the external malleolus and the Achilles tendon, passing forward ½ inch below the malleolus, and extending for 4½ to 5 inches. The skin flap is dissected upward. The extensors of the little toes are pulled forward, and the peroneal tendons pulled backward. An incision similar to the first is made through the ligamentous and capsular covering of the ankle-joint to expose the tibio-astragalar joint. The foot is dislocated inwardly, care being taken not to use too much force, which might cause fracture of the tibial tip. The surfaces of the tibia, fibula, and astragalus are then denuded of cartilage. This procedure is facilitated by using the

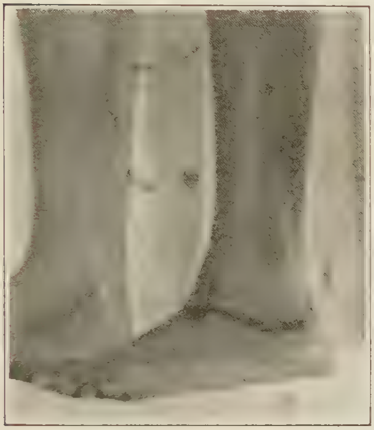
FIG 132

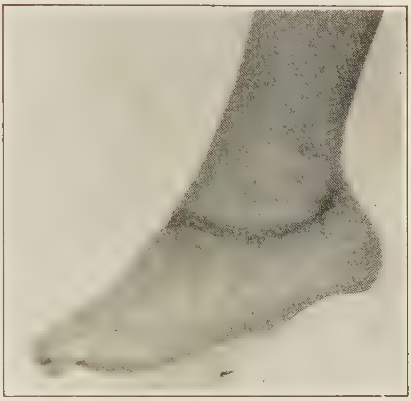
FIG. 133

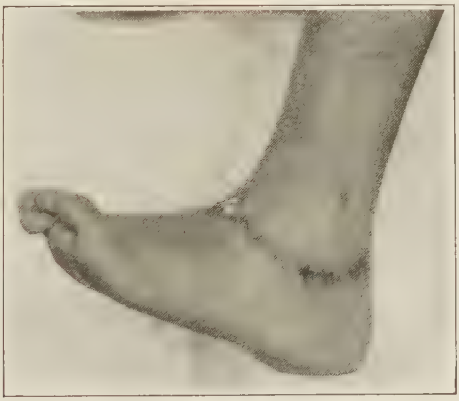
FIG. 134

FIGS. 132, 133, and 134.—Case XXI. D. B. *Cavus deformity.* Photographs taken three months after subastragalar arthrodesis.

chisels devised by Jones, of Liverpool. The dislocation is reduced by bringing the denuded bone surfaces together. This is not easy to do, because the atrophy of the bone and the removal of cartilage leave the astragalus wabbly in its new articulation. To obtain approximation of the opposing surfaces, the fibular tip is fractured by subcutaneous osteotomy and pressed against the astragalus (Fig. 136). Approximation may also be obtained by splitting the astragalus longitudinally nearly to its base and spreading the halves (Fig. 137). The latter method has the advantage of maintaining the tibia and fibula in normal relation.

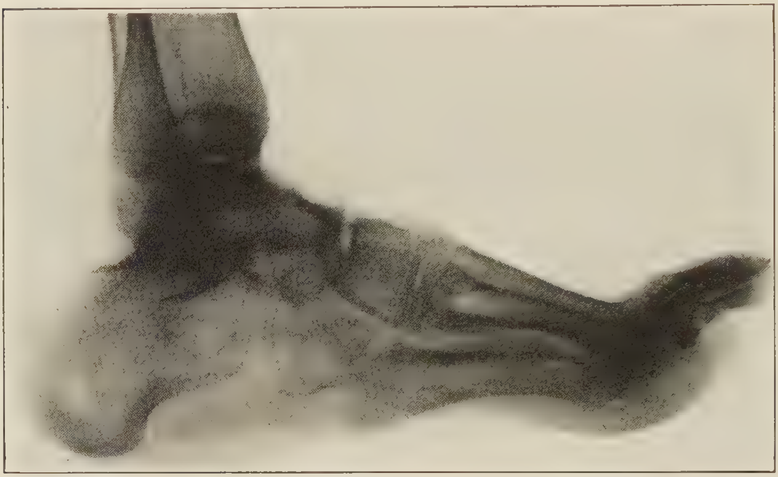

Fig. 135.—Case XXI. D. B. *Cavus deformity.* Lateral radiogram taken eight months after subastragalar arthrodesis.

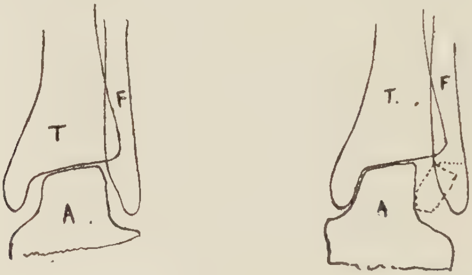

Fig. 136.—*Arthrodesis of the ankle-joint.* Approximation secured by fracture of the fibular tip.

After the bones have been adjusted, the capsule is sewed with chromic catgut, and the skin with catgut or silkworm gut. A sterile dressing is applied. The leg from the toes to above the knees is then encased in a plaster bandage, with the foot held slightly below a right angle and the knee slightly flexed. Plaster bandages are worn over a period of six months, the first one remaining on three to six weeks, and new ones being applied at intervals of four to six weeks.

Results.—The results of this operation are very satisfactory; a firm bony ankylosis is obtained, which gives the patient a good functional foot. Occasionally, a fibrous ankylosis results, due undoubtedly to improper approximation of the astragalus to the tibia and fibula.

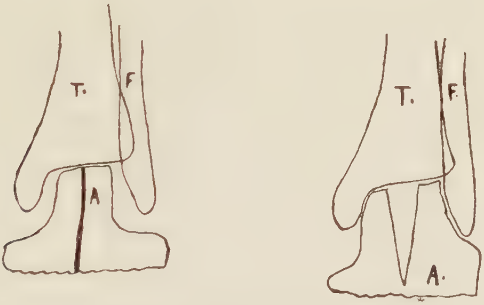

FIG. 137.—*Arthrodesis of the ankle-joint.* Approximation by splitting the astragalus.

CASE XXII.—F. M., aged fifteen years.

Complaint: Paralysis of the left leg.

Duration: Three years.

Cause: Infantile paralysis.

Physical Examination: The ankle was flail. There was marked atrophy of the calf.

Treatment: July 2, 1906. The regular arthrodesis of the ankle joint was performed.

Result: April 12, 1909. The foot was in good position, and there was no motion at the ankle-joint. There was free motion in the mid-tarsal joints. The patient limped slightly because of shortening of the affected leg, and a compensating lift for the shoe was ordered (Fig. 138).

Arthrodesis of the Hip.—This operation is required in only very rare cases.

Operative Technique.—An antero-lateral incision running parallel to the femur is made on the outer aspect of the thigh, beginning at the anterior-superior spine and curving outward 3 inches below the trochanter. The skin and fat are retracted. The greater trochanter is then exposed by incising the fascia. The trochanter is detached from the femur with a saw, and the gluteus medius lifted up to expose the upper capsule of the joint. The capsule is incised by a curved incision running parallel to the acetabulum, and the femoral head dislocated through the wound. The acetabulum and the head are denuded of cartilage. The head is then replaced in the acetabulum.

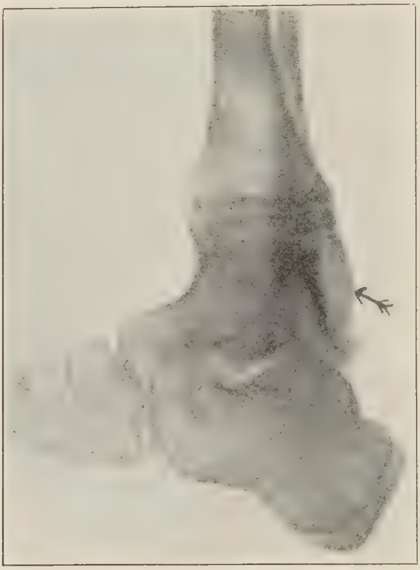

Fig. 138.—Case XXII. F. M. *Flail-ankle.* Lateral radiogram showing an absolute bony union three years after arthrodesis of the ankle-joint.

The capsule is sewed with chromic gut, the greater trochanter is attached to the femur, the fascia is closed with interrupted heavy catgut, and the skin with continuous catgut. The hip is encased in a plaster spica in the position of extension and abduction. The plaster is worn for about four months. Walking may be allowed about the second or third week if there is no pain.

Arthrodesis of the Shoulder-joint.—Provided the muscles that elevate the scapula are strong, fixation of the shoulder-joint in functional position, that is, in about 60 to 70 degrees abduction, with the arm placed 10 to 15 degrees forward of the mid-line, is a most satisfactory operation. Following the arthrodesis, the scapula becomes hypermobile. If the trapezius and the serratus magnus are preserved, good motion as to abduction may be expected, and if the rhomboids and pectoralis minor are present, good forward and backward movement is possible.

Operative Technique. An incision running parallel to the humerus is made over the deltoid muscle, beginning above the acromion, and extending to a point 4 or 5 inches below the shoulder-joint. The skin and fascia are retracted. A similar incision is made through the deltoid muscle into the deltoid bursa, and through the fascia covering the acromion. The muscle and covering of the acromion are retracted. The biceps tendon is lifted and retracted. The capsule of the shoulder-joint and a little of the periosteum and cartilage over the greater tuberosity are incised longitudinally, and elevated. The cartilage under the acromion is then removed to make a shelf with which the greater tuberosity is to come in contact. A piece of bone is removed from the greater tuberosity, so that when the arm is abducted to an angle of 55 degrees, the greater tuberosity will be in perfect apposition with the acromion and clavicular shelf. The cartilage is removed, in so far as possible, from the head of the humerus and from the glenoid cavity. An assistant holds the arm in an abducted position while the operator, using strong chromic gut sutures, sews the periosteal layer, previously removed from the greater tuberosity, over the acromion and clavicular end. The muscle and fascia are sewed over the same region, and the skin is closed with continuous catgut.

After the wound has been sutured, a plaster bandage is applied over the shoulder, chest, and arms as far down as the wrist. The plaster is worn for a period of four months.

In the end-result of this operation, a slight prominence at the inferior angle of the scapula is noticeable when the arm is at the side. Little objection should be raised to this slight deformity in view of the increase in function gained by the operation.

CASE XXIII.—J. R., aged eighteen years.

Complaint: Paralysis of the left arm.

Duration: Fourteen years.

Cause: Infantile paralysis.

Physical Examination: The arm was held adducted to the side. There was no power in the deltoid, supraspinatus, or biceps. The muscles of the scapula were definitely atrophied. Slight internal and external rotation was possible. There was fair power in the triceps. The muscles of the forearm and hand were intact, but atrophied (Fig. 139).

Treatment: June 27, 1924. An arthrodesis of the shoulder was performed. Plaster was applied with the arm 20 degrees forward of the mid-line and in abduction of 70 degrees.

Result: October 8, 1924. Firm union between the humerus and the shoulder had occurred. Photographs (Figs. 140 and 141), taken nine months after operation, show an excellent functional shoulder. The patient can raise his hand to his face and head. He even has played baseball.

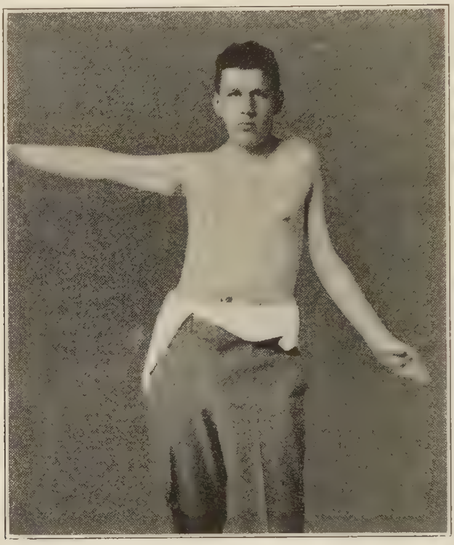

FIG. 139.—Case XXIII. J. R. *Paralysis of the shoulder muscles.* Showing left arm before operation.

Fusion Operation of the Spine (Hibbs[26]).—The fusion operation, as it has been worked out by Hibbs, is based upon the method that Nature adopts to check the progress of scoliotic curves. It is not unusual to find natural fusion of the lateral processes, or of the laminæ, at the time of operation.

The age most satisfactory for spine fusion is young adolescence, but arthrodesis may be necessary before the age of fifteen years in order to safeguard the child against the development of deformity. The failure of fusion to hold in young children is not always due to faulty technique, as is commonly supposed, but to the fact that bony union does not take place, for in young children fusion occurs more slowly between the vertebral bodies than in the small joints.

Before the fusion operation is performed, the deformity should be corrected as much as possible. This step is very important, because a rounded back is likely to follow a fusion operation, when deformity has not been corrected previously to the maximum degree. Reduction is obtained most effectively by placing the patient on a Whitman frame and applying traction. The amount of force that a child can stand without serious discomfort is surprising; sometimes 60 to 100 pounds are borne comfortably. The degree of improvement is ascertained regularly by radiograms, and the surgeon must judge when the limit of reduction has been reached.

Operative Technique.—The back is prepared on the evening before operation, by shaving, and scrubbing its entire surface with green soap and water. Further preparation is carried out just before operation by applying first a tincture of iodine and then alcohol.

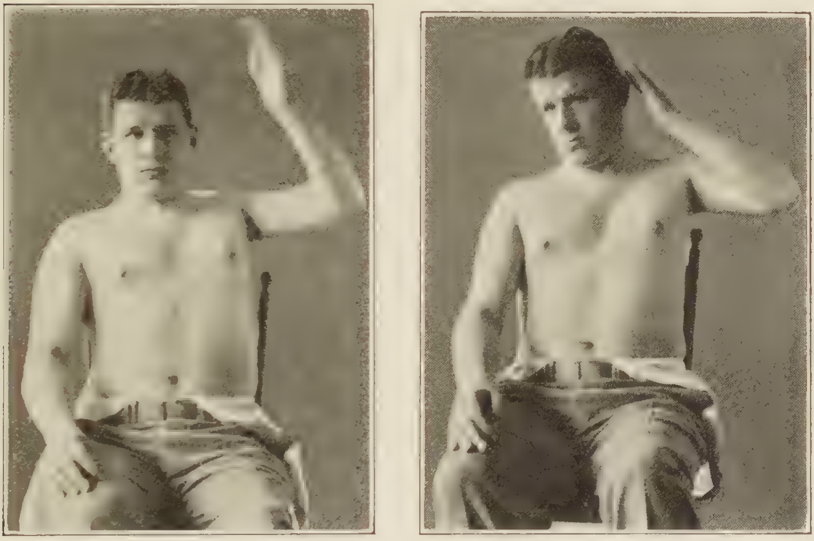

FIG. 140. FIG. 141.

FIGS. 140 and 141.—Case XXIII. J. R. *Paralysis of the shoulder muscles.* Function of the shoulder nine months after operation.

An incision is made through the skin and subcutaneous tissue, from above downward, to expose the tips of the spinous processes of the vertebræ to be fused. The periosteum over the tips of the spinous processes, and the interspinous ligament, in turn, are split and pushed away from the processes. With a periosteal elevator the tips are bared of periosteum for about one-half their length. Small packs of gauze are inserted to prevent oozing. The dissection is then carried farther forward upon each vertebra, beginning at the upper one, until the spinous processes, the posterior surfaces of the laminæ, and the bases of the transverse processes are bared, thereby exposing the ligamentum subflava attached to the margins of the laminæ and the lateral articulations. The insertion of packs to keep the operative field dry is very important.

The ligament is removed from the laminæ with a curette, and the articulations of the lateral processes are destroyed to establish bone contact at these points. This procedure is carried out on each vertebra in turn, care being taken to insert packs.

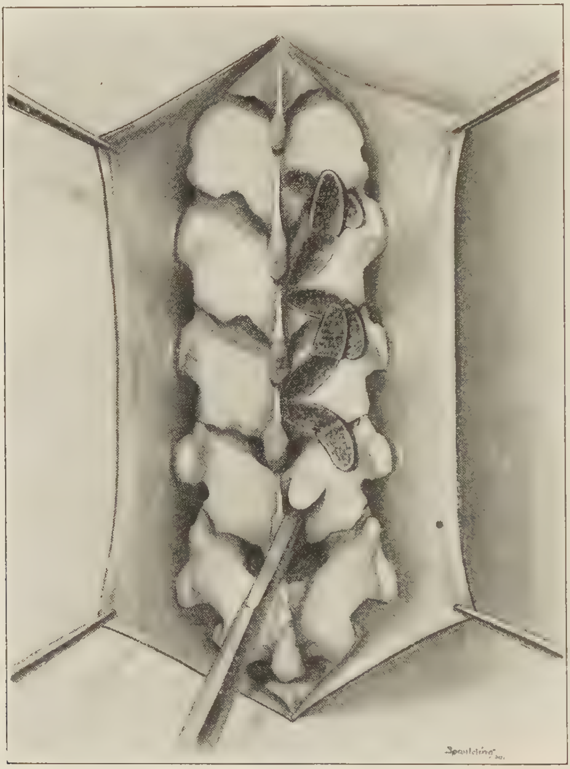

Fig. 142.—*Fusion operation of the spine.* The periosteum has been dissected from the spinous processes and laminæ. The ligamentum subflava has been removed, and the articulations of the lateral processes destroyed. Contact between the laminæ is being established by elevating a piece of bone, and turning it onto the laminæ below and above.

Contact between the laminæ is secured by elevating a piece of bone from each lamina, one-half its size in thickness and one-half its width, and turning the free end of the piece of bone from above onto the lamina below, and the free end of the piece from the lamina below up to the lamina above (Fig. 142). Another means of obtaining contact is to turn the piece of bone from each lamina from above downward onto the lamina below, on one side, and on the opposite side to turn the piece of bone upward.

With a bone forceps, each spinous process is cut and fractured, and its tip brought in contact with the bare bone of the vertebra below (Fig. 143). The spinous process of the lowest vertebra should be turned up to bring about contact with the one above. It may be advisable to split the spinous processes of the lumbar region, which are wide, and to turn one-half up and one-half down.

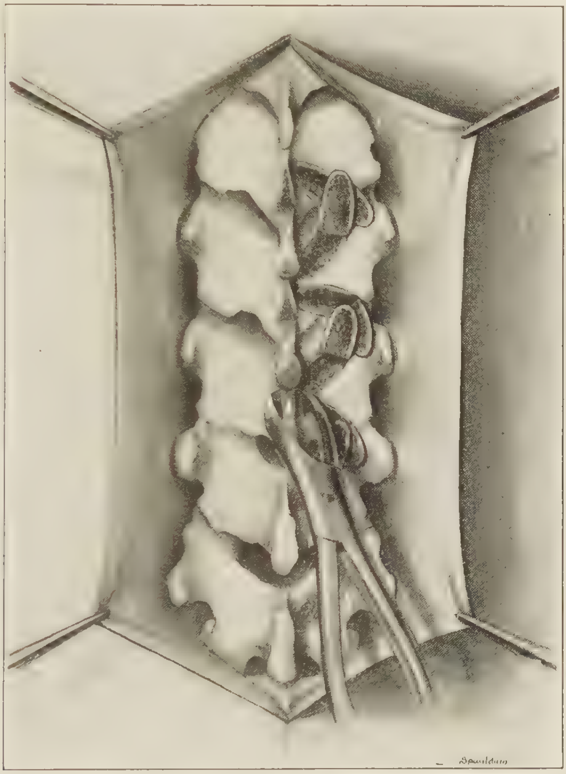

FIG. 143.—*Fusion operation of the spine.* Contact between the laminæ and the lateral processes has been established. The spinous processes are being fractured and their tips brought in contact with the vertebræ below.

The periosteum and ligament, which have been pushed to each side and lie in a practically unbroken sheet, are brought together in the middle with interrupted sutures of ten-day chromic catgut.

The subcutaneous tissue is closed with a continuous suture of plain catgut. The skin wound is closed with sutures of silk. Sterile dressings are applied.

We have now a tube of periosteum, whose blood supply is practically undisturbed, filled with live, healthy bone, in which contact has been established at five points of each vertebra, at the lateral articulations, at the laminæ, and at the spinous processes.

The dissection should be carried out very carefully, as the process of fusion depends upon thoroughness. The dissection may be made in practically a dry field, if it is done subperiosteally, and packs are used freely.

The area to be fused varies according to the extent of the curve, but it should always include one vertebra above and one below the involvement. Hibbs also calls attention to the advisability of fusing the fifth lumbar to the sacrum in cases of dorsal scoliosis with a marked lateral tilt of the fifth lumbar. If this is not done, the articulation of the fifth lumbar with the sacrum is subjected to even greater strain than in the normal spine, with the result that pain occurs after fusion of the vertebræ, especially in the adult. Ossification of the fifth lumbar to the sacrum transfers the lumbo-sacral juncture to the articulation of the fifth and fourth lumbar vertebræ, thereby establishing greater stability.

The operation involves very little danger. In a series of 59 cases operated by Hibbs, there were only 2 deaths, 1 patient dying of pneumonia, and the other of acidosis.

Postoperative Treatment.—For ten days to two weeks the patient lies at rest. At the end of this period, when the wound has healed, the patient rests on a Whitman frame, or in a plaster shell, or in bed with a hyperextension roll below the scapulæ and a fracture board under the mattress. After six weeks a light plaster jacket is applied, and the patient allowed to get up. When the plaster jacket is removed, a leather support should be worn. It is very important to continue supportive treatment for at least six months.

Case XXIV.—J. G., aged seven years.

Complaint: Scoliosis. The patient stood with a right dorsal and left lumbar rotary scoliosis of the rigid type.

Treatment: As much correction as possible was obtained by the application of Abbott jackets and traction (Fig. 144).

October 30, 1923. A fusion operation was performed.

Result: June 13, 1924. The fusion was solid and holding well (Fig. 145). In *February, 1926,* function was considerably improved.

CASE XXV.—W. L., aged sixteen months.

Complaint: Paralysis.

Duration: Six days.

Cause: Infantile paralysis.

Physical Examination: October 17, 1916. The patient lay quiet, without moving the arms or legs. Both arms, both legs, and the back were paralyzed. There was rigidity of the neck muscles. The tenderness was marked. The plantar reflexes were normal.

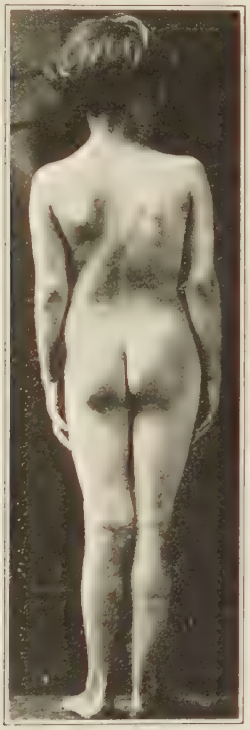

FIG. 144. —Case XXIV. J. G. *Scoliosis.* Before spine fusion, showing degree of correction secured by Abbott jacket.

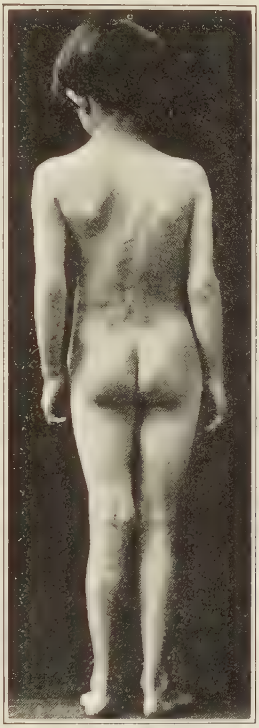

FIG. 145.—Case XXIV. J. G. *Scoliosis.* Photograph taken eight months after fusion operation.

Treatment: When the tenderness subsided, a double spica was applied, with the thighs in slight abduction, the knees straight, and the feet at right angles to the legs. The spica extended as high up as the chest.

Bronchitis and diphtheria interfered with the treatment of this patient. Paralysis of the thoracic muscles prevented his expelling secretions from his chest. He was fretful and nauseated. It was

February of the following year before his general condition began to improve.

In examination at that time *(February, 1917)*, the patient could move his arms well. The biceps and triceps were in good condition, and the deltoids were regenerating. He had left lateral scoliosis, which was being treated.

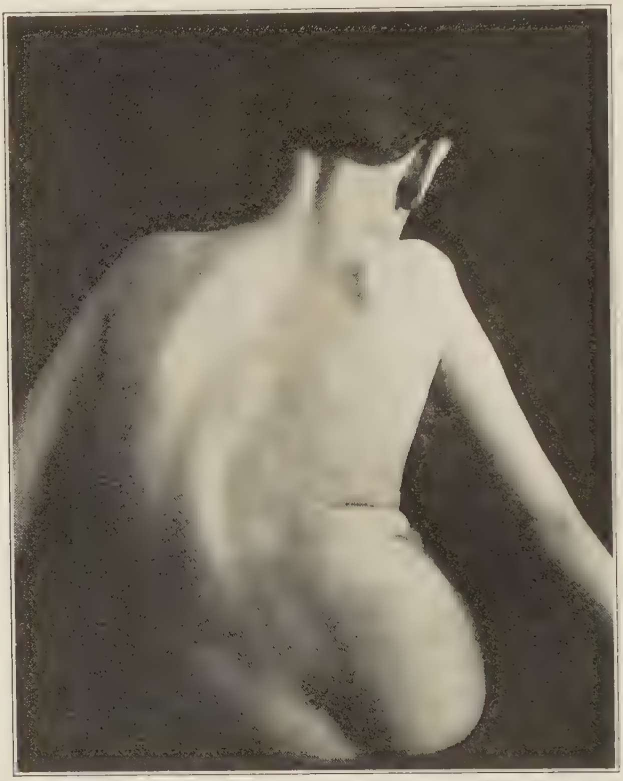

FIG. 146.—Case XXV. W. L. *Scoliosis.* Before spine fusion, showing inability to sit up without support.

During the next five years, from *February, 1917,* to *February, 1922,* the patient wore braces for the back condition. Examination, *February, 1922,* showed lumbar scoliosis and a very marked tilting of the pelvis. The patient could not sit up without support (Fig. 146). In the right leg, the iliopsoas and quadriceps had good power. All muscles in the foot, with the exception of the inner group, had power. In the left leg, there was no power in the iliopsoas or in the quadriceps, and the knee was slightly contracted. The left foot was in the position of calcaneovalgus.

February, 1922. A fusion operation was performed on the spine.

Result: February, 1926, four years after operation, the boy can sit without supporting himself (Figs. 147 and 148). Further development of deformity has been prevented by fusion.

Fusion Operation of the Spine (Kleinberg[27]).—This operation may be done in severe deformities in which it is impossible to denude the posterior arches sufficiently for a Hibbs' operation.

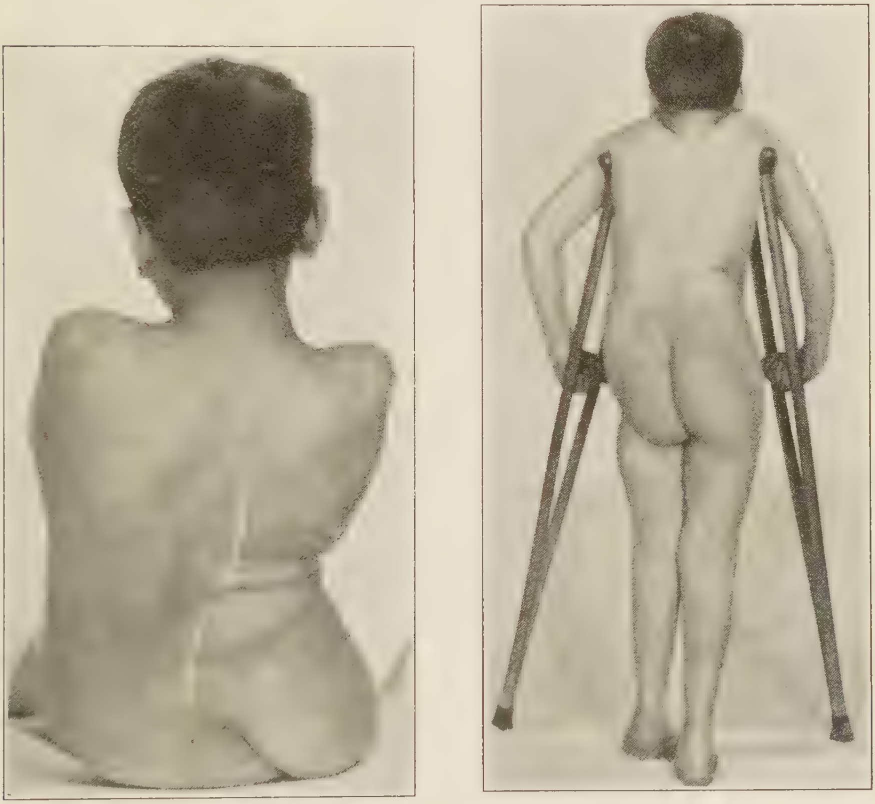

FIG. 147. FIG. 148.

FIGS. 147 and 148.—Case XXV. W. L. *Scoliosis* Photograph taken four years after spine fusion. The patient can now sit up without supporting himself.

Deformity is corrected as much as possible before operation, the same traction method being used as in the treatment preliminary to a Hibbs' fusion operation.

Preparation for Operation.—The preparation begins three days before operation. The traction is released. The diet is controlled carefully to avoid postoperative acidosis. On the first day, the back is shaved, scrubbed with soap and water, and a sterile dressing applied. On the second day, the back is again scrubbed, and a dressing applied. On the third day, the back is painted with 3.5 per cent tincture of iodine, this being done in the morning if the patient is to be operated in the afternoon, and at night, if the operation is to be done on the fourth morning. The dressing applied at this time is not removed until the operation is begun.

Operative Technique.—This operation combines the methods of Hibbs, Albee, and Forbes. The graft of bone to be used is beef bone which has been prepared very carefully. It is cut the proper size, and transverse cuts are made in it to assist vascularization.

A vertical incision is made over the area to be fused. The spinous processes are exposed. The periosteum of the spinous processes is incised and elevated from the underlying bone. The dissection of the periosteum is carried out until the posterior arches of the vertebræ are exposed. In severe cases it is difficult to obtain complete denudation of these arches.

The small articulations between the vertebræ are scarified. Small pieces of bone are elevated from the laminæ and laid across the interlaminar space. The spinous processes of the two upper and two lower vertebræ are split as in the Albee operation. The graft is placed in the wound in contact with the laminæ and transverse processes on the concave side of the curve. The ends of the graft are embedded between the split segments of the spinous processes. The rest of the spinous processes are split horizontally, each into three or more segments (Forbes). These are spread out laterally, and some turned down over the graft and others onto the convex side of the curve.

The periosteum and muscles are sewed with a single row of interrupted kangaroo tendon sutures. The deep fascia, subcutaneous tissue, and skin are sewed in separate layers with catgut.

The extent of the fusion will depend upon the type of curve being treated. In a single curve, all the vertebræ involved in the curve are fused. In a double curve, the section showing the primary curve is operated. In cases in which it is impossible to determine which curve is primary, the area in which the deformity is most marked is operated. In compound curves, the dorsal segment is usually the most prominent, and since the motion in this region normally is limited, fusion does not interfere greatly with the movement of the back. At least eight or twelve vertebræ should be fused. If necessary to fuse more, the operation should be performed in two stages, the dorsal or lumbar vertebræ being fused first, and the other vertebræ later.

Postoperative Treatment.—The patient is put to bed with the foot of the bed elevated, and blankets and extra heat applied. At the end of a week, the patient is again placed on the frame. A few days later, traction is applied to hold correction during the fusing process. The patient remains on the frame for eight weeks. At the end of this period, a light plaster jacket is applied, and weight-bearing allowed. Jackets are worn for at least six months, and then the wearing of a corset is advisable.

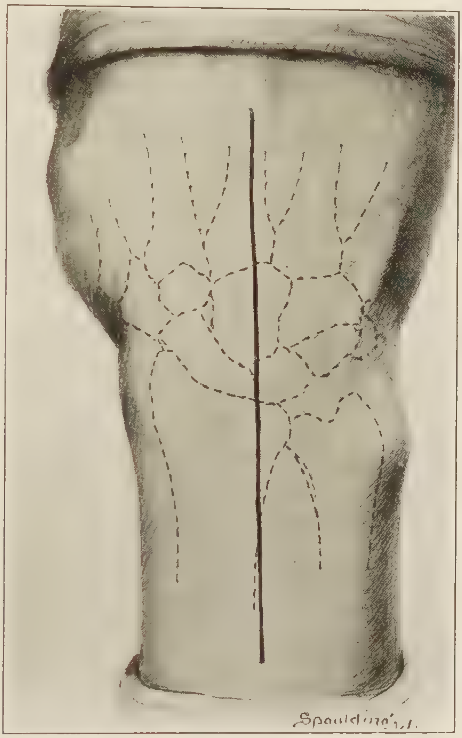

FIG. 149.—*Arthrodesis of the wrist-joint.* Line of incision.

Arthrodesis of the Wrist-joint.—**Operative Technique.**—A posterior longitudinal incision 5 inches long is made over the wrist-joint (Fig. 149). The tendons are exposed and retracted. The posterior wrist-joint is exposed, and the cartilage removed from the scaphoid, cuboid, radius, and ulna (Fig. 150). The parts are then brought into apposition and fixed by bone sutures. The wound is closed in the usual manner, and a plaster bandage applied with the wrist in the position of hyperextension. The plaster is worn for at least three months. Some form of protective apparatus, such as a leather wristlet, is worn for another three months.

Cuneiform Osteotomy for the Correction of Bony Deformity.—In varus, valgus, and cavus deformities of long-standing, the removal of a wedge may be necessary to secure correction. The direction of the apex of the wedge of necessity will vary according to the correction to be made.

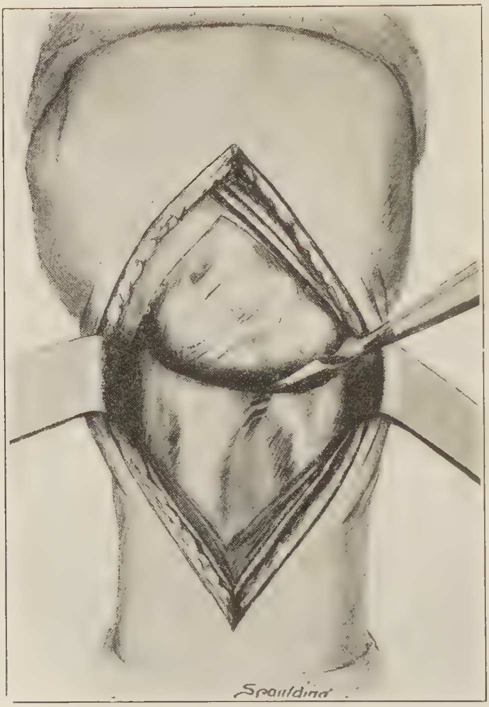

FIG. 150.—*Arthrodesis of the wrist-joint.* Cutting out of joint line with curved chisel, and removal of cartilage.

Operative Technique in Varus Deformity.—Before operation, the foot must be manipulated to correct deformity as much as possible.

A curved anterior incision is made through the skin and fascia over the body of the astragalus (Fig. 151). The tendons are retracted. An incision is made through the ligaments over the body and neck of the astragalus, sufficient to allow exposure of the neck of the astragalus. A V-shaped piece of bone, with its base pointing outward, is then removed from the body of the astragalus (Fig. 152). The amount of bone removed will depend upon the judgment of

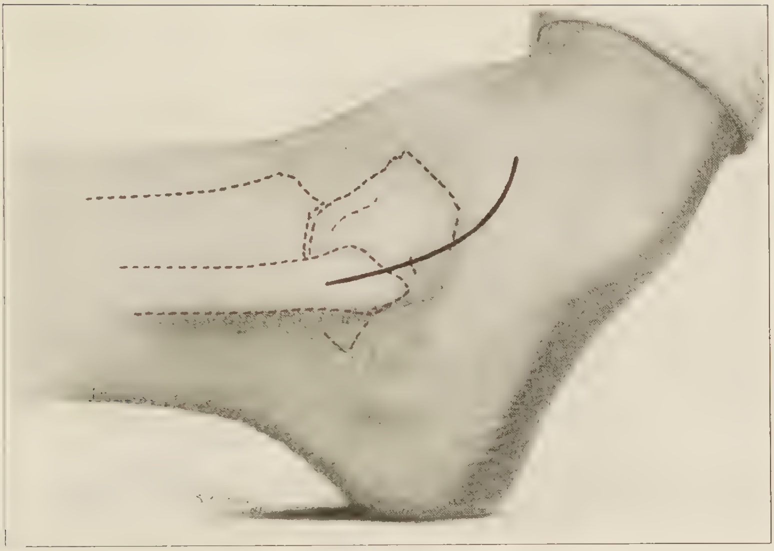

FIG. 151.—*Cuneiform osteotomy for the correction of talipes varus.* Incision over the body of the astragalus.

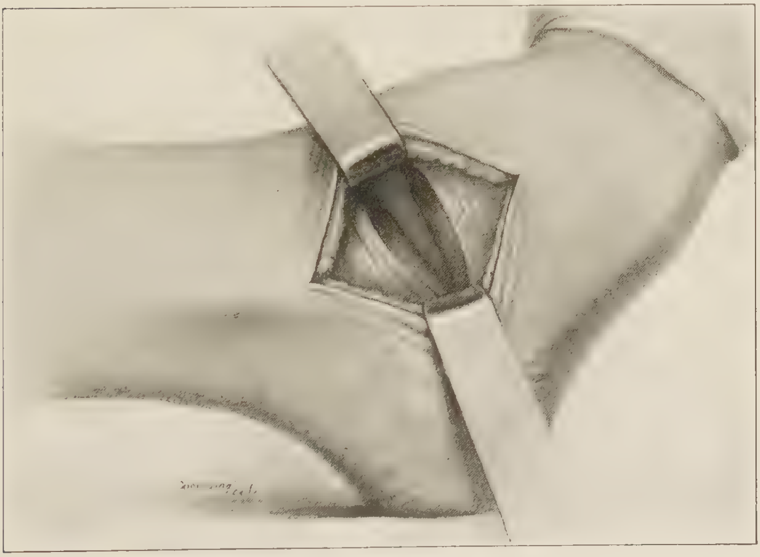

FIG. 152.—*Cuneiform osteotomy for the correction of talipes varus.* Removal of a V-shaped wedge from the astragalus.

the surgeon, but it must be sufficient to overcorrect the deformity. It is also important for the operator to work in front of the astragalotibial joint, to obviate any difficulty in that joint from the formation of callus during convalescence. The gap is then closed, and the ligaments are reunited with chromic catgut. During this process the foot should be held in valgus and slight dorsal flexion.

After the closure of the skin wound, a plaster bandage is applied with the foot in valgus. The plaster remains on for ten weeks.

It may be necessary to supplement this operation by a transplantation of the anterior tibial tendon into the cuboid.

Operative Technique in Cavus Deformity.—In cavus deformity, the wedge, with its base toward the dorsal surface of the foot, is removed from the top of the astragalus. The foot is then manipulated into dorsal flexion, and the gap closed. A plaster bandage is applied and worn for six to eight weeks.

Operation for Calcaneocavus (Jones[28]).—This operation is indicated in a foot so flail that no use can be made of the muscles. It gives the best results when used in children more than eight or nine years of age.

Operative Technique.—The operation is performed in two stages, with an interval of two weeks between each procedure. "In the first stage the plantar fascia is divided if contracted, and stretched by hand or wrench. An incision is made down to the bone about 3 inches long on the inner side of the foot, the center coming opposite the angle of convexity. With a periosteum elevator the soft structures are separated from the tarsus above, and below from the inner to the outer side. A transtarsal V-shaped section of bone is removed, and if there is valgoid deformity the section is wider on the inner side than the outer side. The wound is sutured, and the cavus deformity obliterated by extending the foot which is now bandaged to the tibia, the calcaneus deformity being apparently much increased, and the foot placed in plaster in exaggerated calcaneus.

"The second stage consists of a longitudinal incision made at the back of the heel, the center being opposite the ankle-joint. The ankle and astragalo-calcanean joints are exposed and arthrodesed, the foot being fixed in slight plantar flexion. If the calf muscles are acting, the astragalo-calcanean joint is alone arthrodesed, and the posterior muscles may be used to reinforce the calf muscles. If the calf muscles do not act, but the peronei and sole muscles do, they may be inserted into a tunnel bored through the os calcis, and the tendo Achillis may be divided and the lower end, longitudinally slit, may be passed through a tunnel bored into the tibia or fibula or both. In this way a very firm short ligament is secured which materially aids the transplantation."

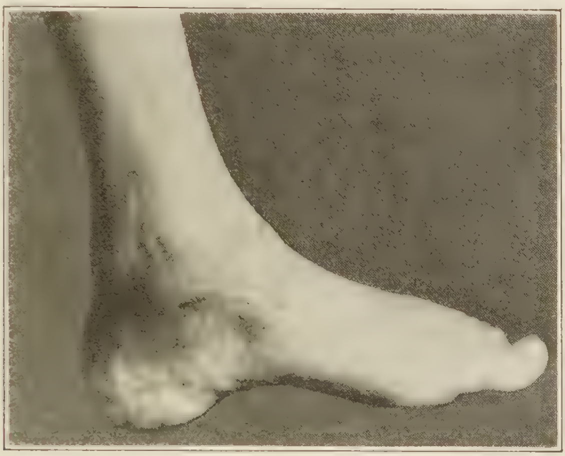

Fig. 153.—Case XXVI. *Calcaneocavus deformity.* Lateral view before operation.

If there is a good deal of lateral movement, Jones performs a subastragalar arthrodesis in addition to this operation. The arthrodesis may be done by enlarging the incision for the removal of the wedge in the forefoot.

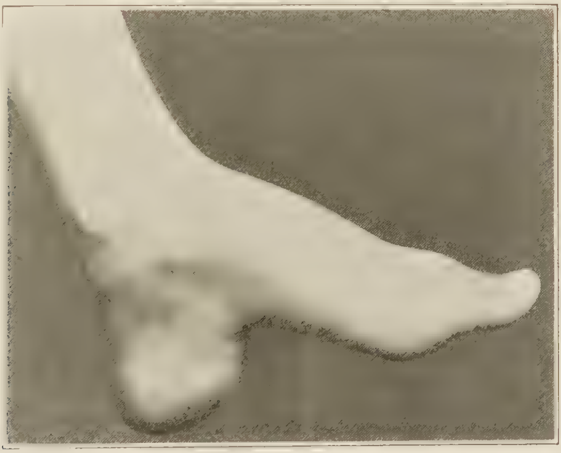

Fig. 154.—Case XXVI. *Calcaneocavus deformity.* Correction following Jones' operation for calcaneocavus. (Courtesy of Sir Robert Jones.)

A case in which a good result was obtained by this operation is illustrated in the photographs (Figs. 153 and 154).

Bone Block Operation for Drop-foot (Campbell[29]).—The object of this operation is to construct a bony process on the superior surface of the os calcis, which will limit plantar flexion by impingement on the posterior surface of the tibia. It is a simple surgical measure and has the advantage of maintaining the rocker-motion of the ankle-joint, as well as conserving the muscle power by eliminating the pull of gravity on the foot.

The procedure is applicable not only to simple drop-foot, but also to cases in which drop-foot is associated with other deformity.

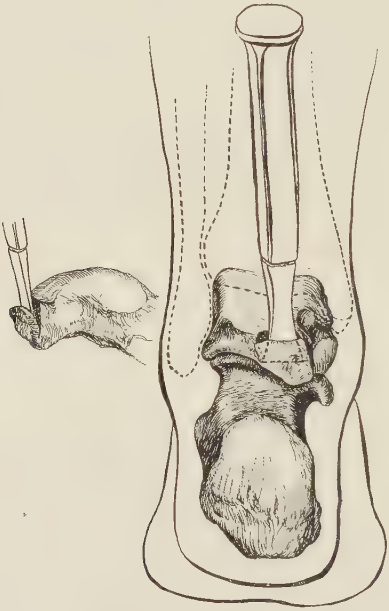

Fig. 155.—*Campbell's operation for correction of drop-foot.* Large piece of bone to be placed on the upper surface of the os calcis.

In equinovarus, the varus is corrected by tarsectomy at the time the bone is removed to form the block. In equinovalgus, a tarsal resection may be done, which will provide bone for transference. In flail-foot, this operation, when used in conjunction with a subastragalar arthrodesis, assures stabilization.

The block operation is not indicated until the child is over eight years of age.

Operative Technique (Figs. 155 and 156).—"The skin incision is made over the Achilles tendon, if this structure is contracted; otherwise to the outer or inner side, as convenient. It should extend from the superior aspect of the tuberosity of the os calcis upward for 3 or 4 inches in a straight line. If the Achilles tendon is contracted, a severance is made by the Z plastic method. A straight incision is next made in the mid-line to the posterior portion of the ankle-joint, retracting inward the tendon of the flexor longus pollicis. With a heavy periosteal elevator, a pyramidal space is cleared, exposing the posterior surface of the tibia, ankle-joint, subastragalar joint and superior surface of the os calcis. The foot is dorsiflexed, bringing into view the posterior extremity of the astragalus, which is removed for from ½ to ¾ inch. A wedge-shaped cavity is excavated on the os calcis, below the posterior extremity of the astragalus and the posterior portion of the articular surface of the tibia. Bone is next transplanted from any portion of the skeleton. As it is usually necessary to enter the forefoot for the purpose of stabilization by arthrodesis, bone is often excised in the process, and can be utilized as transplants. Spongy bone is believed more naturally to adapt itself than dense bone from the tibia, though a tibial transplant was successfully employed in one case and has been recently advocated by Leo Mayer. Transplants from the femur, when the case is complicated by genu valgum, and from the ilium, in transferring the crest in hip flexion contractures, have proved efficient. If bone is excised from the forefoot, it is denuded of cartilage, and a large piece about ½ by 1 inch in diameter is selected and placed in the cavity prepared on the superior surface of the os calcis, about which small particles are arranged in a pyramid. The cartilage removed is placed in mosaic fashion to cover the posterior, inner and outer

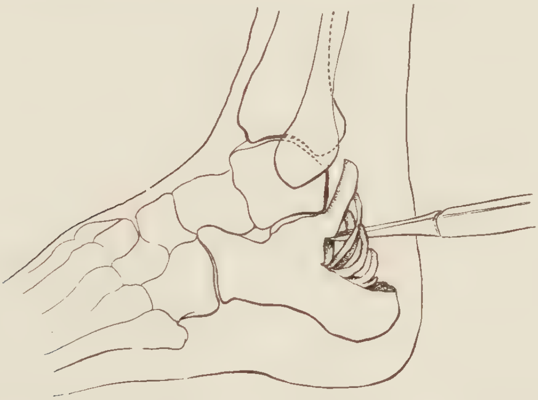

FIG. 156.—*Campbell's operation for correction of drop-foot.* Diagram showing bone block in position approximate to and within the ankle-joint.

aspects of the pyramid, to prevent adhesion to surrounding structures; this step, however, is by no means essential, as the results are as good when bone is obtained elsewhere, free of cartilage. The soft parts are sutured snugly to retain the transplants *in situ* the Achilles tendon is united, if severance is required, and the fascia and skin are closed. The foot is then held by a plaster cast at an angle of 90 degrees with the leg. Overcorrection is especially avoided, as it is preferable to secure a slight degree of plantar flexion, in order to avoid flat sole or heel walking. At the end of eight weeks, the cast is removed and a roentgenogram made, which usually demonstrates proliferation. A sequence of roentgenograms exhibits a gradual organization of bone, until there is a solid mass continuous with and forming a part of the os calcis and often fusing the os calcis to the astragalus, stabilizing the subastragalar joint. . . The technique may be modified, when the forefoot is stable, by turning forward the upper third of the os calcis, when, incidentally, it is possible to do a subastragalar arthrodesis, or even the horizontal section of Davis, with backward displacement of the foot by the posterior route. It is also possible to transfer particles of bone from the mid-tarsal region through the subastragalar joint to the superior surface of the os calcis, in performing a triple arthrodesis, but the block cannot be so accurately placed as when the posterior incision is employed.

"Great care must be taken to place and to retain the bone block approximate to the ankle-joint and the posterior surface of the tibia. Displacement is avoided by an apparatus that prevents plantar flexion. Consolidation is usually complete by the end of six months, when the apparatus may be discarded."

Results.—Campbell has employed this operation in 135 cases. Of 62 of these cases, examined from six months to three years after operation, there were 58 cases in which the plantar flexion was restricted satisfactorily by a definite bony impingement. Three failures were attributed to errors in technique, and one to absorption of the transplant.

Comment.—The writer has had no experience with this operation, but the results which have been obtained by Campbell are very promising, and there is every indication that the bone block will act as a permanent means of preventing foot-drop. There is little possibility of the development of a painful joint, since the bone block acts as a check when weight is off the foot, at which time there is no irritation at the points of impingement.

The following case, with photographs and radiograms, was lent to the writer by Dr. Campbell.

CASE XXVII.—L. M., aged nineteen years.

History: At the age of ten months this patient had an acute illness, which was followed by paralysis of the right leg. She was unable to walk until six years old. She then began to use crutches, and continued to walk with them until nineteen years of age.

Examination: R. A. M., $30\frac{1}{4}$; L. A. M., 33. Right hip in socket. No contracture. Flail. Right knee, quadriceps 0. Internal hamstrings 0. External hamstrings 25 per cent.

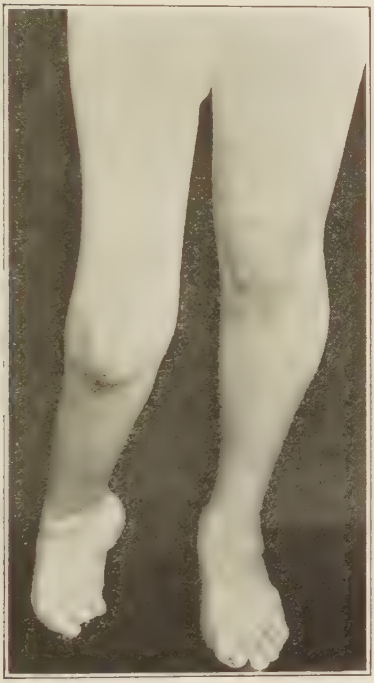

FIG. 157.—Case XXVII. L. M. *Talipes equinovarus and cavus.* Before bone-block operation.

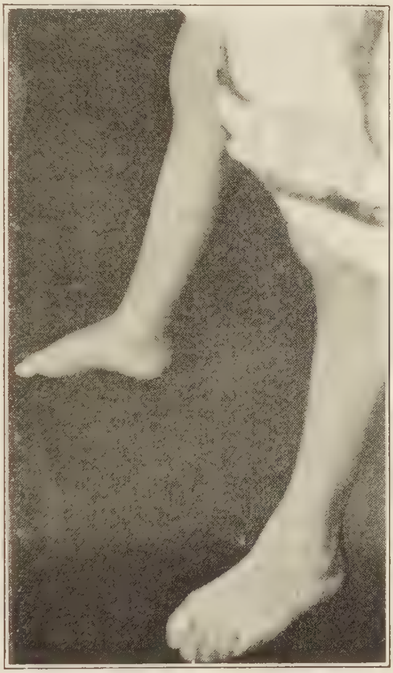

FIG. 158.—Case XXVII. L. M. *Talipes equinovarus and cavus.* Four months after bone-block operation. (Courtesy of Dr. Campbell.)

Foot, equinovarus and cavus. Gastrocnemius 50 per cent. Posterior tibial 25 per cent. Anterior tibial 0. Toe flexion 25 per cent. Toe extension 25 per cent. Peroneals 0. Left hip and knee normal. Left foot planus. Muscle power normal, except for anterior tibial 25 per cent (Fig. 157).

Treatment: A subcutaneous Steindler operation and an arthrodesis of the astragalo-scaphoid and calcaneo-cuboid joints were performed. Sufficient bone was removed to correct the varus deformity. The tendo Achillis was lengthened, and the drop-foot operation performed.

November 9, 1925. A new cast extending from the toes to the groin was applied, and Martin's knee extension apparatus used.

December 9, 1925. A manual osteotomy of the femur and displacement of the epiphysis were performed to correct the flexion deformity of the right knee. An osteotomy of the tibia and fibula also was done, and the foot and leg manipulated to correct the outward rotation.

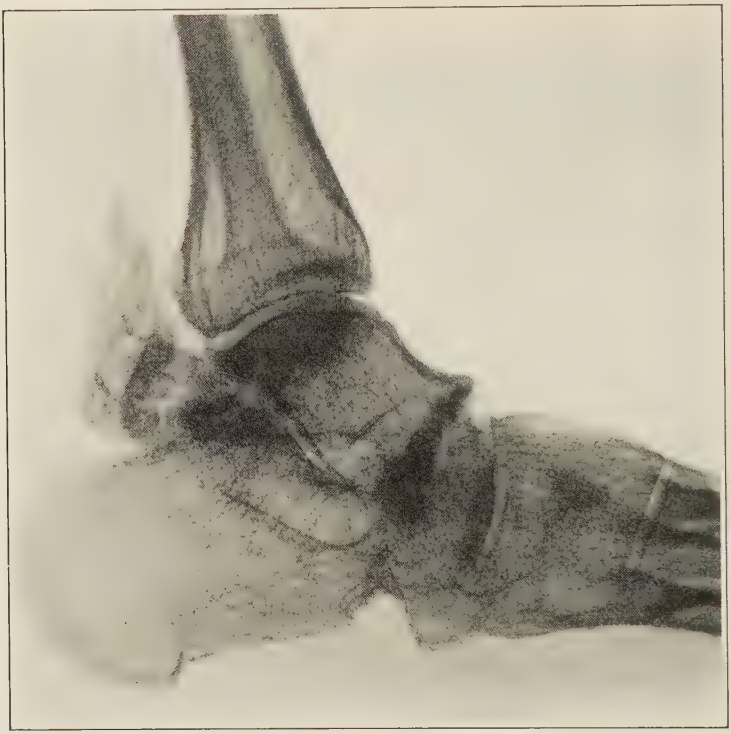

Fig. 159.—Case XXVII. L. M. *Talipes equinovarus and cavus.* Radiogram four months after bone-block operation.

February 1, 1926. A drop-ring knee brace with a night splint was fitted.

Result: February 10, 1926. The illustration (Fig. 158) and radiogram (Fig. 159) show the satisfactory result of the bone-block four months after operation.

Osteotomy of the Femur for Genu Valgum.—Operative Technique.—A 2½-inch incision is made on the outer aspect of the femur just above the tuberosity. The skin and fat are strongly retracted, and at the same time the biceps tendon is drawn back by the lower retractor. The femur is exposed by blunt dissection, and osteotomized half-way across as near the condyle as possible. The femur is fractured and correction made. The wound is closed with continuous catgut.

A plaster bandage is applied from the toes to the groin, with the leg held in overcorrection. Weight-bearing is allowed in about six weeks. The plaster is removed in nine weeks.

Osteotomy of the Tibia and Fibula for Eversion of the Lower Leg. —Operative Technique. A 2-inch incision is made over the tibia at the juncture of its middle and lower thirds. The skin and fascia are retracted. A longitudinal incision is made in the periosteum, and this is retracted. A transverse osteotomy is done through the tibia, and the periosteum is sewed tightly. A subcutaneous osteotomy is then done through the fibula, opposite the point of osteotomy in the tibia. The deformity is corrected by strong internal rotation. A plaster bandage is applied from the toes to the groin. Weight-bearing is allowed in two weeks. The plaster is removed in six to eight weeks.

CASE XXVIII.—F. F., aged twelve years.

Complaint: Paralysis of the left leg.

Duration: Long standing.

Cause: Infantile paralysis.

Physical Examination: This patient entered the hospital at the age of nine years, with the left leg everted and the foot in the position of equinus. There was no power of extension in the leg, and no power of flexion or extension in the foot. The thigh and calf muscles were atrophied, and the left leg was about 2 inches shorter than the right (Fig. 160).

Treatment: February 13, 1913. An arthrodesis of the ankle-joint was performed.

July 20, 1916. On account of the eversion of the lower leg, a rotation osteotomy of the tibia was performed.

Result: August 30, 1919, three years after correction of the eversion, the patient could walk with the foot directly ahead, and the function was thereby considerably increased (Fig. 161).

There was a tendency to knock-knee, which has since been corrected by an osteotomy of the femur.

Tibio-astragaloid Arthroresis *(Putti[30]).*—This operation, which is designed to limit joint motion, may be used to stabilize a knee in which extension is weak or lost entirely as the result of paralysis of the quadriceps. When lateral instability is present, a subastragalar arthrodesis is required in addition to the arthroresis. An arthroresis should not be performed in children under the age of eight years.

Operative Technique.—A longitudinal incision is made in front of the ankle-joint. The extensors are retracted, exposing the tibia and the anterior surface of the astragalus. A transverse trough is made in the body of the astragalus by cutting it at about the middle and elevating the upper part. A block of bone, 3 cm. by 2 cm. is removed from the anterior surface of the tibia and implanted in the trough. Thus a ledge is formed, which limits dorsiflexion above an angle of 110 degrees. The foot is immobilized in slight plantar flexion for three or four months.

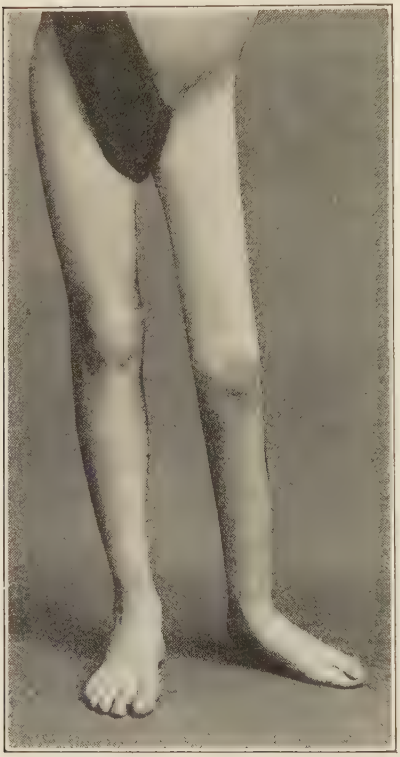

Fig. 160.

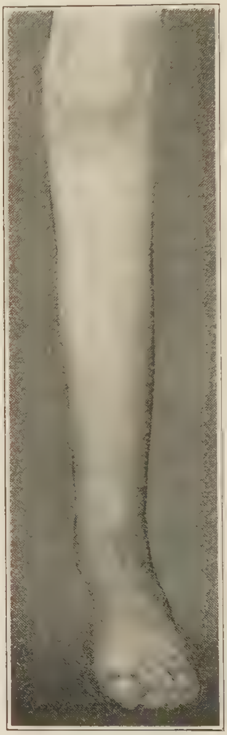

Fig. 161.

Fig. 160.—Case XXVIII. F. F. *Eversion of the lower leg.* Before operative treatment, showing equino-valgus of the foot and eversion of the lower leg.

Fig. 161.—Case XXVIII. F. F. *Eversion of the lower leg.* Result, three years after osteotomy of the tibia.

Results.—Mezzari[30] reported on 23 cases operated by this method. The arthroresis had been done from two months to four years previously. In the majority of cases, the implant became a part of the astragalus. In general, the functional results were excellent.

REDUCTION OF PARALYTIC DISLOCATED HIP.

Closed Operation. -**Operative Technique.**—The patient lies on his back, with the thigh hanging just off the table. Assistants hold the opposite leg on the table and grip the pelvis firmly. The operator, using the knee and thigh for leverage, slowly forces the leg into abduction. The head usually slips readily into the old acetabulum. A plaster spica is then applied with the hip in the position of abduction. A spica is worn six months, the abduction being reduced each time the bandage is changed. An abduction hip splint is used when the final spica is discarded.

Open Operation.—In old-standing cases, reduction by the closed method is difficult because of the extreme relaxation of the posterior and superior capsule, the contraction of the inferior capsule, the shallowness of the acetabulum, and the flatness of the femoral head.

Operative Technique.—A two-day preparation of the operative field is made, including the knee-joint, the inner and posterior parts of the thigh to the groin, and the section from the anterior mid-line to the posterior mid-line well above the crest of the ilium.

After the patient has been draped properly, a towel is placed over the thigh and indentations made, beginning at the anterior-superior spine and running downward, in a slightly curved manner, to the outer part of the thigh. An incision 6 to 8 inches long is made. The skin edges are wiped with alcohol and clamped off with towels. A longitudinal incision is made through the fascia, care being taken not to carry it too low, in order to avoid the muscular branches and the nerve leading to the vastus externus. The external crural artery, which usually appears in the lower part of the wound, should be clamped off and tied. The rectus muscle and the sartorius are defined and retracted to the inner side, and the tensor vaginæ femoris and the gluteus medius and maximus are defined and retracted to the outer side. All tissue is then freed from the capsule by dissection.

An incision, running parallel to the shaft of the femur, is made through the capsule and enlarged up and down with heavy scissors. The joint cavity is explored with the index finger. The femoral head is usually found high and posterior. The teres ligament is located and will be found to lead to the old acetabulum. The inferior capsule is cleared away from the acetabulum with a chisel, and the cavity cleaned of all soft tissue. Any obstacles to reduction are overcome. The hour-glass-shaped constriction, usually found at the point where the teres ligament comes out by the upper edge of the acetabulum, is carefully divided. Occasionally, it is necessary to sever the attachments of the pectinei on the inner side of the femur. Sutures are then placed in both sides of the capsule, as it is difficult to define it when reduction has been made.

The reduction of the head into the acetabulum is now attained by a combination of abduction and rotation movements. The writer's congenital hip skid is placed under the back of the head, the lip extending just inside the superior rim of the acetabulum. The operator then pushes on the trochanter, at the same time lifting the skid, while an assistant brings the leg into abduction, strong inward rotation, and hyperextension. Considerable inversion is required to force the rotated head into proper apposition to the acetabulum. When the head reaches the acetabulum, it snaps into place and is locked there by further abduction and inward rotations.

A curved shelf of bone, about 1½ inches wide and 4 to 5 inches long, is raised from the ilium, as close to the old acetabulum as possible and curving about the head with its base near the old hip-joint. The fragment is bent out so that it protrudes at a right angle to the ilium. This shelf of bone may be reënforced by a bone graft and chips of bone. The process thus formed acts mechanically as an obstacle to the recurrence of dislocation.

The capsule is then overlapped, and the chromic gut sutures, which had been placed in it previously, are tied tightly. The muscles fall back in place, a catgut suture usually being required to prevent a blind pocket. The fascia is closed with continuous chromic catgut, and the skin with continuous catgut. Plaster is applied from the breastline to the toes, with the hip in abduction and inward rotation, and the knee flexed. This spica remains on for eight weeks. At the end of this period, the knee is extended, and a new spica applied with the hip in 15 to 20 degrees abduction and marked inward rotation. The patient is then allowed to bear weight.

DIAMOND-SHAPE OPERATION FOR FLAIL-ELBOW (Jones).

Operative Technique.—A diamond-shaped incision is made, beginning at the center of the biceps muscle, extending to the anterior surfaces of the condyles, and continuing to a point on the forearm opposite to the starting point. The diamond-shaped flap of skin and fascia thus formed is removed, and the lower and upper surfaces of the edges of the wound united with catgut.

A plaster bandage is applied with the arm flexed at 45 degrees beyond a right angle. The plaster is worn for six to eight weeks.

This operation occasionally is performed in combination with arthrodesis of the shoulder.

Results.—While the immediate results of this procedure are excellent, the benefits are often not permanent because of the stretching of the skin and fascia. In some cases, power returns in the biceps when the arm has been held in flexion, and in other cases, in which the biceps is totally paralyzed, the supinator may take on the power of flexion. The return of power in the biceps or the action of the supinator longus helps materially in producing flexion.

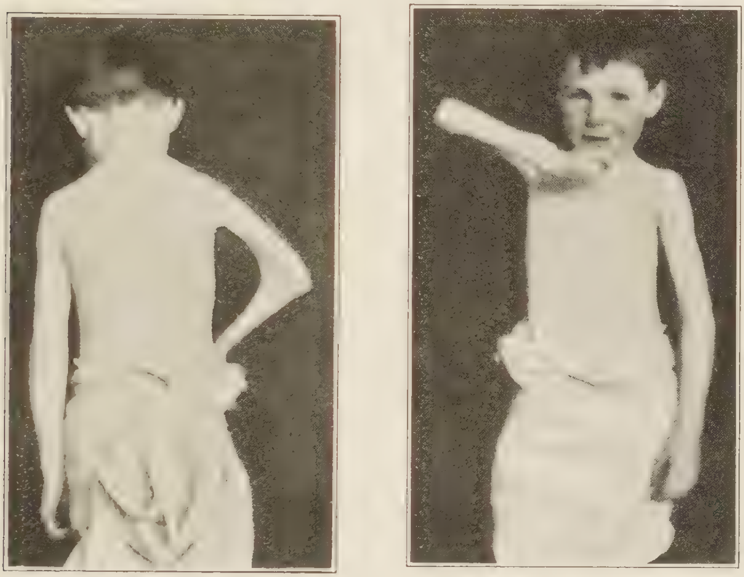

Fig. 162. Fig. 163.

Figs. 162 and 163.—Case XXIX. T. R. *Paralysis of the right shoulder and arm.* Photographs showing result one year after diamond-flap operation and arthrodesis of the shoulder.

Case XXIX.—T. R., aged six years.

Complaint: Patient cannot use his right hand.

Duration: Five years.

Cause: Infantile paralysis. When the child was one year old, he suffered an attack of poliomyelitis, which left the right shoulder and arm paralyzed.

Physical Examination: All the muscles in the shoulder region were paralyzed. There was good power in the triceps and biceps. Power was present in the forearm muscles, but it was weak.

Treatment: August 11, 1914. An arthrodesis of the right shoulder and a skin plastic operation of the elbow were performed.

Result: September 8, 1914. The patient could hold objects in his hand. One year later, *September 21, 1915,* he was able to get his hand to his mouth (Figs. 162 and 163). There was also firm bony union at the shoulder.

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CHAPTER XIV.

APPARATUS AND PLASTER.

From the time when Heine received wide recognition for his success in treating paralytic cases by the use of mechanical appliances, apparatus has been used extensively. The modern types of apparatus present a great improvement over the early crude forms. At the present time, owing to our progress in orthopedic surgery, the prescribing of braces and splints is defined more carefully than formerly. Apparatus now is seldom used as a permanent measure of relief, since it is possible to dispense with appliances by operation. Today, a brace is used to hold a limb temporarily in correct position and to furnish support to weakened muscles. The writer uses apparatus only in the late convalescent stage, for he has found plaster bandages more efficient in holding the limb in position during the early stage.

Essentials of Apparatus. -There are certain principles that must be observed in the construction of all apparatus used for paralytic conditions. A brace must be simple and light in construction; the type of appliance prescribed must meet the requirements of the given case; and finally, the brace must fit accurately.

The simple and light brace is the most efficient. Steel or aluminum may be used in its construction. Heavy and complicated apparatus adds to the disability by materially impeding the use of the limb. Mechanical appliances of the Hessing type are little used.

Only one who has had a thorough training in the construction of the different forms of appliances is qualified to prescribe a brace or splint. Braces work in certain fixed lines and planes, and may be equipped with free, limited, or stop-joints. The selection of the type of joint depends upon the requirements of the given case.

The fitting of braces is a science in itself and requires considerable patience. Appliances should fit snugly, but should not be tight or cause pressure. Constricting straps or bands which interfere with proper circulation of the limb should not be used. Buckles and straps should be adjusted so that they do not bind and rub.

As the child grows, or as the condition improves, the apparatus will need alteration and adjustment. It is necessary to inspect the braces from time to time to make sure that they are efficient and that they are not exerting undue pressure.

Uses of Apparatus. -Mechanical appliances are used for several purposes:

To Prevent Deformity.—Apparatus is of value in preventing deformity and maintaining the limb in the position most favorable to the recovery of the weakened muscles. As soon as the plaster shell which is used to hold the weakened extremity in overcorrection is removed, some form of apparatus usually is required to prevent the unopposed muscle pull. The brace or splint is worn until the weakened part has recovered sufficiently to resume its function, or until operative interference is indicated.

To Maintain the Part in Position Following Correction of Deformity.—When the plaster bandage, which has held the limb in position following correction is removed, apparatus may be used as a further means of maintaining the part in an overcorrected position. Some European surgeons, including Sir Robert Jones of Liverpool, use mechanical apparatus with a remarkable degree of skill to hold the limb in position immediately following the forcible correction of deformity. In America, we rely on a plaster bandage for this purpose and use apparatus in late convalescence.

To Correct Deformity.—Apparatus may be of help in bringing leverage to bear for the correction of mild deformity. For instance, in beginning contraction of the knee, the use of a caliper brace with an anterior leather cap which exerts pressure on the knee will correct the mild flexion.

To Support Weakened or Paralyzed Limbs.—In the presence of muscle weakness, apparatus prevents fatigue by maintaining the weakened part in a position of rest. In weakness of the quadriceps, for example, the patient will stretch and tire the muscle groups unless apparatus is used. In other cases, apparatus may support weakened muscles so that walking is possible.

Mechanical appliances are also used for the fixation of flail joints to enable the patient to be ambulatory.

Apparatus may be used as a permanent substitute for a paralyzed muscle group, when no benefit is to be expected from surgery.

To Compensate for Shortening.—It is imperative to compensate for shortening, in order to prevent the development of secondary deformities. Some form of raised shoe may be used for this purpose.

Types of Apparatus.—Many forms of apparatus have been devised to meet the needs of paralytic conditions. Included in this section is a description of those most commonly used by the writer.

Thomas Heel.—The inner side of the ordinary heel of a shoe is extended outward gradually from the back, until the extension at the anterior end is $\frac{3}{8}$ inch in width and has been carried $\frac{1}{2}$ inch forward of the normal heel. A lift is inserted, measuring $\frac{1}{4}$ inch on the anterior part of the heel, $\frac{1}{8}$ inch on the posterior, and beveled at the center line.

Raise on Heel and Sole.—A raise measuring from $\frac{1}{8}$ to $\frac{1}{4}$ inch and beveled to the center line is placed on the inner side of the heel. A similar raise $\frac{1}{8}$ inch in height is added to the inner side of the sole. The raise may be placed on the outside of the heel and sole, or inserted as a wedge between the lifts of the heel and sole.

Reverse Raise on Heel and Sole.—This raise, similar to that described above, is added to the outer side of the shoe.

Whitman Brace.—This brace is made of steel and carefully molded to a plaster model of the patient's foot. It has three unbalanced points which, when weight is borne, roll the plate outward, throwing the foot into varus.

In extreme valgus, the Whitman brace is used in conjunction with a raised heel and sole, or with a valgus shoe.

Moderate Square and Long Spring Plates.—The moderate square plate consists of a steel sole plate extending from the forward-bearing surface of the heel to the metatarsal heads, with a flange on the outer side. The main features of the brace are the dome in its forward part, which raises the anterior arch, and the roll on the inner side of the plate, which holds the posterior part of the foot in position. The plate is covered with leather, and a leather strip extends back under the heel.

This brace is fitted to a plaster model of the patient's foot. The anterior and posterior arches are raised to the height required for correction.

The long spring plate is similar to the moderate square, except that the steel is extended under the heel, and the outer flange may be omitted.

Valgus Brace.—The valgus brace consists of an arched sole plate, extending under the sole of the foot from the metatarsal heads to the back of the heel, with an outer flange on which are three buckles. The plate itself is arched to as near the normal degree as possible. On the outer side of the brace an upright extends to just below the knee-joint. The upright bulges out at the ankle, is equipped with a stop-joint at this point, and ends in a broad flange, from which a strap passes about the calf (Fig. 164).

The following measurements should be taken to ensure proper fitting of the brace:

Length of sole.
Breadth of ball.
Breadth of heel.
Length of upright.
Height from sole to ankle-joint.
Circumference of calf.
Outer upright with right-angle stop-joint.

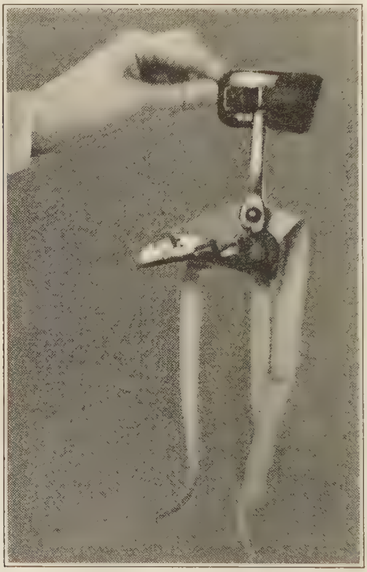

Fig. 164.—Valgus brace.

In the application of the brace, the straps around the foot are first adjusted. Any degree of correction may be obtained by bending the upright to secure leverage. This apparatus may be used night and day for one to six months after the removal of plaster. Later in convalescence, the brace may be worn only at night, and a valgus shoe may be substituted during the day.

Valgus Shoe.—An upright is added to the outer side of a shoe, extending from the sole to just above the top of the shoe. A band and strap hold the upright in position. A T-strap, attached to the inner side of the shoe at the heel and buckled around the outside of the upright, holds the foot in a corrected position. A stop-joint, limiting motion at or above a right angle, is usually applied to the valgus shoe.

The upright may be incorporated between the leather and the lining of the shoe. The T-strap may be attached inside the shoe, and the end of the strap brought out under the tongue and fastened to a peg on the outer upright.

A raised heel and sole should be used in conjunction with the valgus shoe.

Varus Brace. -The varus brace is similar to the valgus brace, except that the upright comes on the inner side of the leg. A reverse raised heel and sole is always used with this brace.

Varus Shoe. The varus shoe is similar to the valgus shoe, except that the upright is on the inner side, and the T-strap and raised heel and sole are on the outer side of the shoe (Fig. 165).

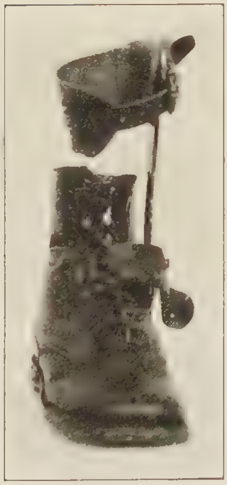

Fig. 165.—Varus shoe.

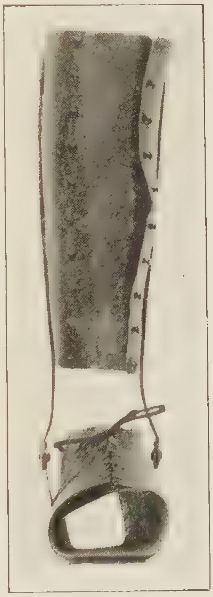

Fig. 166.—Arthrodesis brace.

Arthrodesis Brace. The arthrodesis brace consists of an arched sole plate to which is attached a single or double upright. An anterior leather cuff is generally used to give additional support to the foot. The upright is usually made without a joint, but if motion is advisable, a stop-joint is applied (Fig. 166).

This brace is made most satisfactorily over a plaster model, but very careful tracings may be used if a model cannot be obtained.

Arthrodesis Shoe.—Two rigid uprights are placed on either side of the shoe and attached above to a calf band. The only measurements necessary for its construction are the length of the uprights, the circumference of the calf, and an antero-posterior tracing of the leg.

Equinus Brace.—The equinus brace consists of a sole plate, to which is attached a single or double upright, equipped with a stop-joint to prevent plantar flexion below 90 degrees. It depends largely upon the mechanical problem to be solved, whether a double or single upright is used. For children, the single upright is advisable, as it is lighter and just as efficient. The brace may be worn inside the shoe.

Equinus Shoe.—This consists of a shoe in which is incorporated a double or single upright, equipped with a stop-joint to prevent plantar flexion below 90 degrees.

Equinovarus Brace and Shoe.—In equinovarus deformity, a stop-joint is used on the varus brace or shoe to prevent the foot from dropping below a right angle.

Equinovalgus Brace and Shoe.—In equinovalgus deformity, a stop-joint is used on the valgus brace or shoe to prevent the foot from dropping below a right angle.

Calcaneus Brace and Shoe.—The calcaneus brace and shoe are similar to the equinus brace and shoe, except that a reverse stop-joint prevents dorsal flexion above 90 degrees.

Calcaneovarus Brace and Shoe.—In calcaneovarus deformity, the varus brace or shoe is equipped with a reverse stop-joint which prevents dorsiflexion of the foot above 90 degrees.

Calcaneovalgus Brace and Shoe.—In calcaneovalgus deformity, the valgus brace or shoe is equipped with a reverse stop-joint which limits dorsiflexion.

Cork Wedge and Raise.—When the amount of shortening is from $\frac{1}{2}$ to 1 inch, compensation is secured by adding a $\frac{1}{4}$ inch raise on the outside of the entire heel on the affected side, and by removing a $\frac{1}{4}$-inch lift from the entire heel of the other shoe. Any further compensation is obtained by inserting in the shoe a cork wedge, which is raised to the desired height at the heel and is tapered to nothing at the ball of the foot.

Cork Heel and Sole.—When the amount of shortening is more than 1 inch, but does not exceed 3 inches, the cork heel and sole is used as a compensatory means. For example, when the shortening amounts to 2 inches, it is compensated in the following manner: On the impaired side, a cork wedge, raised $\frac{1}{2}$ inch at the heel, $\frac{1}{4}$ inch at the sole and tapered to nothing at the toe, is inserted into the shoe. On the outside of the shoe, a cork wedge, raised $1\frac{1}{4}$ inches at the heel, 1 inch at the sole, and $\frac{1}{4}$ inch at the toe, is inserted between the lifts of the sole and heel. The shoe is raised less at the toe to allow the patient to roll forward in weight-bearing.

Extension Shoe.—In severe shortening, an extension shoe is required. This is actually a shoe within a shoe.

Cabot Posterior Wire Leg Splint.—This splint is quadrilateral in shape, and is made of $\frac{1}{4}$-inch wire. The rod is bent upward at the heel to form a right-angle foot-piece, 8 to 10 inches in length and corresponding in breadth to the width of the foot. The upper frame is modeled to conform to the surfaces of the calf and thigh. This frame of wire is bandaged and padded. It is retained in position by adhesive plaster, or by straps and buckles applied near the upper limit of the splint, above the ankle-joint, and around the foot near the great toe-joint.

Leather Leglet.—The leather leglet is efficient in treatment of paralysis about the knee. It is made from a plaster model of the leg, taken from the thigh to the ankle. The leglet is laced in front. Support is furnished by steels placed at the sides and by thigh and calf bands. If the knee is to be kept immobilized, a posterior steel is placed in the leather. When motion at the knee is desired, the leather about the knee is cut away, and a joint, allowing the proper degree of motion, is incorporated in the lateral steels (Fig. 167).

Caliper Brace.—The simple caliper brace consists of two uprights which extend from a socket in the heel of the shoe to within $\frac{1}{2}$ inch of the groin, where they terminate in a posterior thigh band with an anterior leather strap. This band is applied at an angle of 45 degrees. The simple caliper brace has a rawhide knee-cap. A narrow leather strap buckles around the ankle to hold the uprights in their sockets. The simple caliper brace allows free ankle-joint motion, except when used in talipes equinus and talipes calcaneus; in equinus deformity, a stop-joint is applied to the shoe to prevent plantar flexion below 90 degrees, and in calcaneus deformity, a reverse stop is applied to the shoe to prevent dorsal flexion above 90 degrees.

If possible, the brace is made from a plaster model of the leg, taken from the toes to the groin. If it is not convenient to take a cast, a tracing of the leg and measurements of the circumference of the thigh, calf, and ankle will be sufficient.

In the presence of moderate flexion deformity at the knee, the knee-cap may be applied with increasing tightness to secure correction.

In the presence of moderate knock-knee deformity, the knee-cap is extended on the inner side, and is narrowed gradually until it passes in the form of a strap around the outer upright. The purpose of this strap is to pull the knee into correct position. In talipes varus and talipes valgus, T-straps, Thomas heels, and raised heels and soles may be used.

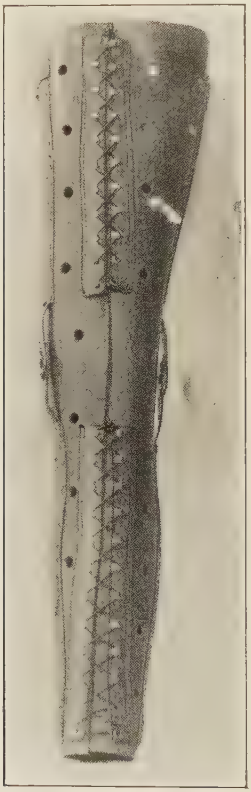

FIG. 167.—Leather leglet.

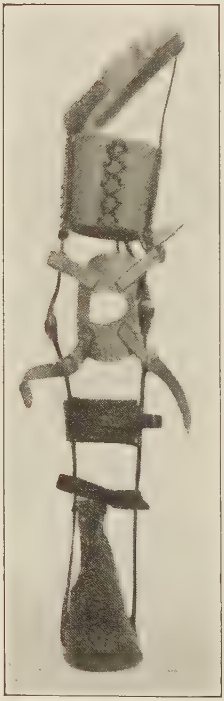

FIG. 168.—Thomas ring caliper.

Thomas Ring Caliper.—The Thomas ring caliper in its simple form resembles the ordinary caliper, except that a padded ring inclined at an angle of 45 degrees takes the place of the posterior band. This ring bears the weight of the pelvis when the leg is too weak to perform this function (Fig. 168).

Various types of joints may be incorporated in the brace, and the choice of a lock depends upon the function required. The drop-catch knee-joint is used most commonly; it ensures stability while the patient is standing, and may be unfastened, while sitting, by pulling up the drop-lock. A spring-lock may be used in place of the drop-catch, but on the whole is is not so satisfactory.

Inversion and eversion deformity of the leg may be controlled by applying straps about the pelvis to rotate the leg outward or inward. Knee-caps and ankle-straps may be used as in the case of the simple caliper.

The method of measurement is the same for the Thomas ring caliper as for the simple brace.

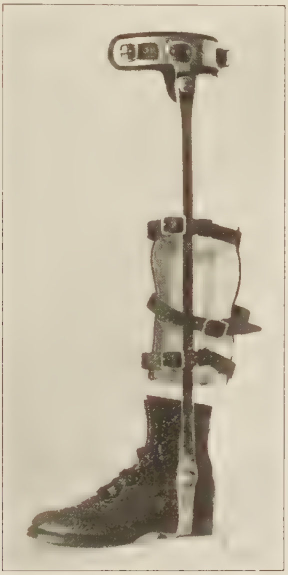

FIG. 169.—Knock-knee brace.

Knock-knee Brace.—The knock-knee brace has a single outer upright extending from the heel of the shoe to the hip. The upright is attached to a short steel band, from which a broad leather strap extends around the pelvis. The brace has free joints at the hip and ankle, and a leather pad on the inner side of the knee to give pressure. The pad is instrumental in correcting the deformity (Fig. 169).

For the proper construction of the brace, the measurement of the circumferences of the hip and knee, a tracing of the leg from the toes to the hip, and dimensions for the knock-knee pad are necessary.

Jones Knee Brace.—The Jones knee brace has two uprights which extend from the calf to the middle thigh and terminate in posterior thigh and calf bands. A knee-cap, similar to that used on the caliper brace, gives anterior support and pressure at the knee. The joint at the knee may be arranged to give the degree of motion desired. In genu recurvatum a stop-joint prevents hyperextension.

Spring Back Brace.—The spring back brace consists of a pelvic band with two uprights which extend to the shoulders. Here they are curved to give the leverage desired. A cross-bar connects the two uprights $\frac{1}{2}$ inch above the level of the axilla. Abdominal support is obtained by means of an apron which straps onto the brace. In slight scoliosis in which a jacket is not indicated, a steel pressure pad may be added to the upright on the side of the rotation (Figs. 170 and 171).

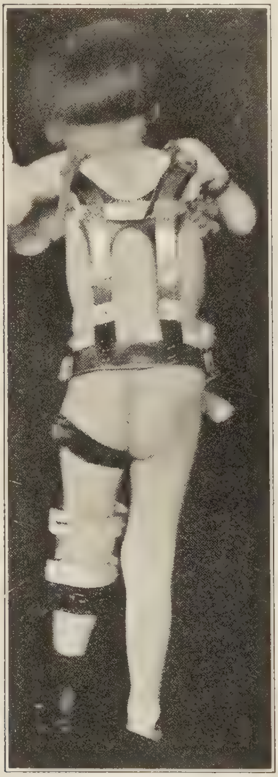

Fig. 170.—Spring back brace.

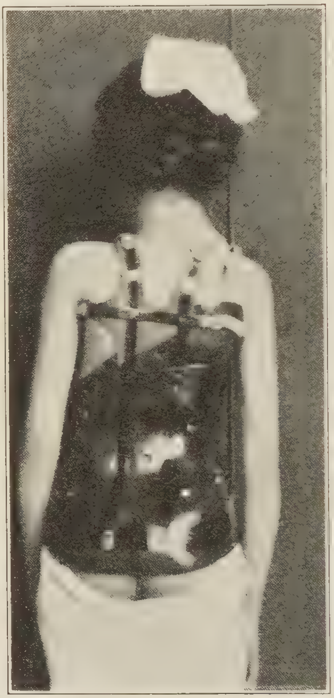

Fig. 171.—Scoliosis jacket with spring back brace incorporated.

Measurements for making this brace are as follows:

Pelvic band: From mid-line between the anterior-superior spine and trochanter on one side to a similar point on the opposite side.

Cross-bar: Distance from axilla to axilla, less 1 inch on either side. The cross-bar is placed on the upright ½ inch above the level of the axilla.

Distance apart at top of uprights: Distance between points halfway between the neck and tip of the shoulder on either side.

Abdominal apron: Height—from pubis to just above umbilicus. Breadth—distance between anterior-superior spines, less ½ inch on either side.

Leather Jacket.—The leather jacket is made over a well-fitting plaster model, taken with the patient in suspension. The cast is filled, convexities are shaved off, and concavities filled in, to the extent that the patient's flexibility warrants. A little plaster is also added over the anterior-superior spine and breasts to avoid pressure on these parts.

After the leather has been modeled to the cast, steels are applied to give the necessary support. In an ordinary jacket, two anterior and two posterior uprights and a pelvic band are used. In scoliosis jackets, a pressure pad is added on the side of the rotation, or a spring back brace is incorporated in the leather.

Whitman Frame. The Whitman frame is made of gas pipe, over which canvas is tightly laced. It should be 6 inches longer than the patient, and the width should correspond to the breadth of the patient at the axillæ. The frame is arched to hold the spine in hyperextension, and the greatest point of the curve, amounting to 25 to 30 degrees, usually comes in the dorsal region at about the middle of the scoliotic curve.

Thomas Collar.—The Thomas collar is made from a tracing of the patient's neck. It is constructed of heavy fiber-board reënforced with light steels and covered with leather. The upper and lower edges of the collar are well padded to ensure comfort. The collar is fastened by two straps at the back of the neck.

Leather Helmet.—A plaster model is made of the upper torso, extending from the chin and occiput down the chest and midway down the back. Leather is modeled to the cast, and well reënforced with steels to hold the head and neck in the position desired. The helmet opens in the back (Fig. 172).

Leather Arm Spica.—The leather arm spica is very carefully molded over a plaster model, including the chest, one-half the back, the shoulder, and the involved arm in the position of abduction. The plaster does not cover the unaffected shoulder, but goes under the arm. The spica is laced up the front and over the affected shoulder. The leather extends from the shoulder in the form of a posterior shell, in which the involved arm is held by means of straps.

Sufficient support is given to the arm and shoulder by means of reënforcing steels.

Abduction Arm Splint.—The abduction arm splint consists of two steel rods across which canvas is stretched. The arm rests on this support in the position of abduction. The splint is held in position by two steel uprights, which are attached to a pelvic band. Numerous straps also serve to hold the splint in place.

Very careful measurements are necessary for the proper construction of this brace.

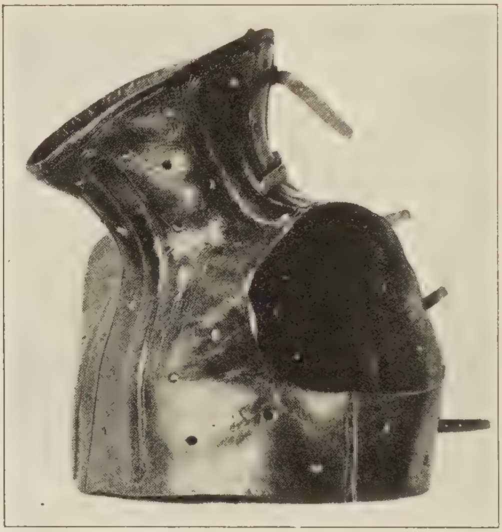

Fig. 172.—Leather helmet.

Pelvic band: Distance from anterior mid-line to posterior midline. The band is curved to fit over the crest of the ilium.

Height of splint: Distance from pelvic band to anterior part of shoulder-joint.

Length of splint: Distance from shoulder to elbow (outside measurements). Distance from elbow to wrist (outside measurements).

Angle wrist is to be cocked up.

Measurements for straps:
- Circumference of pelvis.
- Circumference of chest.
- Circumference of upper arm.
- Circumference of upper forearm.
- Circumference of wrist.

Leather Armlet.—To ensure a properly fitting leather armlet, a cast must be taken of the arm from just below the shoulder to the wrist, with the elbow at a right angle. The armlet is laced on the upper surface. Steels are placed on the outer and inner sides of the leather to maintain the arm in the position desired. If motion in flexion is advisable, a stop-joint is applied at the elbow of the brace to prevent motion below a right angle.

Wrist Hyperextension Splint.—This is a light splint, made of aluminum and covered with leather. It extends well up the forearm and is cocked up at the wrist to fit into the palm. A broad strap at the wrist and a narrow one at the forearm hold the splint in position (Fig. 173).

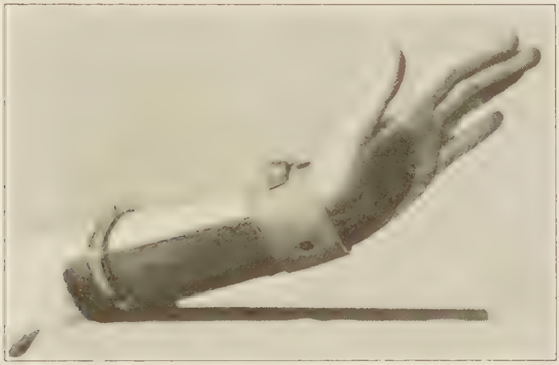

FIG. 173.—Wrist hyperextension splint.

The measurements necessary for its construction are: A tracing of the forearm and palm and the circumferences of the wrist and upper forearm.

Hand Hyperextension Splint. The splint is made of aluminum. It extends half-way up the forearm and includes the whole hand, fingers, and thumb. It is hyperextended at the wrist the required degree. A broad strap at the wrist, a narrow strap around the palm, and tapes at the fingers and thumb hold the splint in place.

For measurements, a tracing of the entire hand and the circumferences of the wrist and palm are required.

Leather Wristlet, With or Without Steel in Palm.—The leather wristlet resembles a glove from which the fingers and thumb have been cut off. It extends to the middle forearm and laces up the back. It may or may not have a steel incorporated into the leather at the palm to maintain hyperextension of the wrist.

The wristlet is molded over a plaster cast.

Hand Stretcher.—This splint, made of light steel, has been devised to secure extension of contracted fingers. The forearm support is similar to that of the hyperextension splint. A steel cross-bar, molded to conform to the contour of the hand, is attached horizontally to the tip of the palm support. A framework, attached to the flanges of the cross-bar, extends out from the hand, its upper part containing four hooks, threaded at one end and held in place by butterfly nuts. An elastic band is attached to each hook and fastened to the finger by means of adhesive tape. Traction is obtained by tightening the screws. More hyperextension at the wrist may be secured by adjusting the screws on the flanges of the cross-bar.

PLASTER.

Plaster is used for several purposes in the treatment of infantile paralysis. In early cases, a plaster bandage is applied to allow the muscles and joints to rest in proper anatomical position. Plaster is used after a manipulative or operative procedure to maintain the affected part in corrected position. Plaster bandages are very useful as models in the construction of apparatus.

The plaster bandage should be light, well-fitted, well-rubbed, smooth, and hard. Before the bandage is applied, the joint is placed in the position desired. The limb is covered with stockinette or layers of sheet-wadding, and all bony prominences are padded with felt. Plaster bandages 4 to 6 inches wide, which have been well soaked, are wound around the affected part, care being taken to rub them carefully during the winding process, and not pull them too tightly.

If the plaster is to be used as a model, it is split and removed before it is thoroughly dry. If the bandage is to remain on, the limb should not be moved until the plaster is perfectly dry. Soon after application, the bandage may be split, and the anterior half removed to allow the limb to receive local therapy.

Objection has been raised to the use of plaster bandages, on the basis that a limb confined in plaster will atrophy. Daily massage and exercise help to prevent atony. When the lower extremities are involved, weight-bearing on a walking-sole helps to eliminate atrophy.

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